This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

LANE
MEDICAL

LIBRARY
Gift
Dr.Jens P.Jensen

MODERN OPHTHALMOLOGY

BALL

MODERN

OPHTHALMOLOGY

A PRACTICAL TREATISE ON THE ANATOMY, PHYSIOLOGY, AND DISEASES OF THE EYE

P. Dennis Brownley

BY

JAMES MOORES BALL, M.D.

PROFESSOR OF OPHTHALMOLOGY IN THE ST. LOUIS COLLEGE OF PHYSICIANS AND SURGEONS

SECOND EDITION

WITH 417 ILLUSTRATIONS IN THE TEXT AND NUMEROUS FIGURES ON 21 COLORED PLATES

PHILADELPHIA

F. A. DAVIS COMPANY

1908

COPYRIGHT, 1904,
COPYRIGHT, 1908,
BY
F. A. DAVIS COMPANY.

[Registered at Stationers' Hall, London, Eng.]

Philadelphia. Pa., U. S. A.
Press of F. A. Davis Company,
1914-16 Cherry Street.

DEDICATED

TO

HERMANN SNELLEN, SR.,

OF UTRECHT, HOLLAND,

AND

CHARLES A. OLIVER,

OF PHILADELPHIA,

IN RECOGNITION OF THEIR VALUABLE CONTRIBUTIONS

TO

MODERN OPHTHALMOLOGY.

PREFACE.

In writing this book it has been the author's aim to produce a work which shall teach, and which shall be valuable alike to the medical student, to the general practitioner, and to the specialist. The author hopes that he has succeeded in correctly representing the present advanced state of ophthalmic science and practice.

Regarding the arrangement of subjects, the author's experience as a teacher of ophthalmology has led him to believe in the advisability of combining embryology, anatomy, physiology, and diseases of the eye within one volume. In the clinical portion of the treatise congenital anomalies, tumors, inflammations and degenerations, injuries, and operations have been considered in the order mentioned. This is believed to be a logical sequence.

The illustrations may possibly be worthy of mention. Many of them are original, and have been made either from the author's dissections or from sections prepared under his direction. The colored pictures, illustrative of external diseases and fundal conditions, are the handiwork of Miss Margaretta Washington, of Philadelphia. While some few have been made from sketches furnished by the author, the majority represent cases examined in the Wills Hospital of Philadelphia, to whose staff the author is under great obligations. The uncolored illustrations have been drawn chiefly by Drs. R. W. Mills and Carl Fisch, of St. Louis. The diagrams are the work of several individuals.

Finding it to be impossible for him to finish the volume within the time-limit granted by the publisher, the author called several well-known ophthalmologists to his aid, all of whom responded courteously. Thus, the subject of "Anomalies of the Muscular Apparatus" was assigned to Dr. William Zentmayer; "Ocular Symptoms of Nervous Diseases," to Dr. Jay C. Knipe; "Diseases of the Orbit," to Dr. William T. Shoemaker;

107930

"Errors of Refraction," to Dr. John T. Krall; "Hygiene of the Eyes," to Dr. Harold G. Goldberg, all of Philadelphia, and "Methods Employed in the Microscopic Examination of the Eye," to Dr. W. E. Fischer, of St. Louis. Drs. Joseph Grindon and A. H. Ohmann-Dumesnil, of St. Louis, have given assistance in the preparation of that part of Chapter V which treats of the dermatologic diseases of the eyelids.

The author desires to thank the F. A. Davis Company for the courteous treatment which he has received. The publishers have spared neither pains nor expense to make this book a worthy representative of its class.

JAMES MOORES BALL.

SAINT LOUIS: 3509 FRANKLIN AVENUE.

CONTENTS.

CHAPTER I.

PAGE

DEVELOPMENT OF THE EYE 1

Development of the eye—The lens—The vitreous humor—The secondary eye-cup and the eye-membranes—The optic nerve—Development of the retina—Accessory organs of the eye.

CHAPTER II.

ANATOMY OF THE EYE 8

The Orbit—Foramina—Interorbital space—Orbital contents—The periosteum—The capsule of Tenon—The orbital muscles—Check ligaments—Nerves of the orbit—Blood-vessels of the orbit—The intermuscular cone of fat—Position of the eyeball—The lacrimal apparatus—The eyelids—Blood-vessels, lymphatics, and nerves of the lids. **Anatomy of the Eyeball**—The fibrous coat (cornea)—Sclera—The vascular coat—Structure of the iris—The ciliary body—The chorioid. **The Optic Nerve, Retina, Lens, and Vitreous**—The optic nerve—The retina—Layers of the retina—Optic part of the retina—Cerebral layer—Neuroepithelial layer—Ciliary and iridal parts of the retina—The optic commissure—Crossing of the optic fibres—Orbital portion of the optic nerve. **Radiations of the Nerves of the Eye**—Optic nerve—Oculomotor nerve—Patheticus nerve—Abducens nerve. **The Crystalline Lens**—Dimensions of the lens—Structure of the lens—The lens-capsule—The zonula. **The Vitreous Body. The Ocular Lymph-Spaces. The Normal Fundus Oculi**—The optic disc—The blood-vessels—Perivascular lymph-channels—The macula lutea—The chorioid—The sclerotic.

CHAPTER III.

PHYSIOLOGY OF VISION 62

Mechanism of the Formation of Images—Refraction—The reduced eye—The visual angle—The retinal image—Accommodation—Optical defects—Functions of the iris—Monocular diplopia. **Ocular Movements and Binocular Vision**—Movements of the eye—Protrusion and retraction movements—Relative ocular movements—Blindness during ocular movements—Binocular single vision—Stereoscopic vision. **Functions of the Retina**—The field of vision—The blind spot—The macula lutea—Direct and indirect vision—The rods and cones in vision—Optograms—Duration of visual sensations—Irradiation—Relation of sensation to the intensity of the stimulus—Time necessary for excitation of the retina by light—Clearness of vision—Inversion of the image—The visual centre—Color-perception—Normal variation in color-perception—Theories of color-perception. **The Ocular Adnexa.**

CHAPTER IV.

EXAMINATION OF THE EYE 85

The case-history. **External Examination**—Eyelids—Eversion of the lids—Ocular conjunctiva—Lacrimal apparatus—The caruncle—The sclerotic—The length of the eye—The cornea—The anterior chamber—The iris—The

PAGE

pupil—The pupillary area—The crystalline lens—The vitreous humor—The ciliary body—The tension of the eye—The orbit—Balance of the ocular muscles—Power of the ocular muscles—Strabismometry—Keratometry—Testing visual acuity—Examination of near vision—Measurement of convergence—Perimetry—Defects in the visual field—Testing the light-sense—Testing the color-sense—The use of mydriatics—The use of miotics. **Ophthalmoscopy**—Description of the ophthalmoscope—Methods of use—The room and light—Position of examiner and patient—Size of the ophthalmoscopic image—Uses of the ophthalmoscope—The fundus-reflex test—Tests for simulated blindness—Localization of foreign bodies.

CHAPTER V.

DISEASES OF THE EYELIDS 158

Congenital Anomalies of the Eyelids—Ablepharia—Coloboma of the lids — Cryptophthalmos — Symblepharon — Ankyloblepharon — Blepharophimosis — Microblepharon — Entropion — Ectropion — Distichiasis — Epicanthus—Congenital ptosis—Fistula of the upper lid—Treatment of congenital anomalies. **Skin Diseases of the Lids**—Erythemata—Urticaria—Eczema of the eyelids—Fissures of the external canthus—Impetigo—Furuncle—Frambesia—Furunculus orientalis—Malignant pustule—Carbuncle—Abscess of the lid—Tarsitis—Erysipelas—Herpes zoster ophthalmicus—Pemphigus—Lichen—Dermatitis—Blastomycetic dermatitis—Perifolliculitis — Congenital hyperkeratosis — Chloasma — Discoloration of the eyelids — Dermoid tumors — Verruca — Cornu cutaneum — Lipoma — Hernia of the orbital fatty tissue — Edema of the lids — Solid edema of the lids — Elephantiasis Arabum—Lentigo—Varicose veins—Nævus pigmentosus—Vitiligo—Xeroderma pigmentosum—Keratosis follicularis—Molluscum epitheliale—Xanthoma—Lupus erythematosus—Tuberculosis—Lupus vulgaris—Syphilis—Leprosy—Keloid—Plexiform neuroma of the eyelid—Fibroma of the eyelid—Nævus vasculosus—Lymphoma of the eyelids—Carcinoma of the lid—Epithelioma—Rodent ulcer—Sarcoma of the eyelid—Adenoma of the Meibomian glands—Noma of the eyelids—Colloid degeneration of the skin—Hyperidrosis—Chromidrosis—Phosphoridrosis and uridrosis—Sudamina—Seborrhea—Miliaria—Milium—Acne rosacea—Asteatosis—Steatoma—Chalazion—Alopecia—Parasitic skin diseases—Favus of the eyelids—Cysticercus of the eyelid—Variola—Vaccinia of the eyelid—Varicella—Scarlatina—Rubeola—Blepharochalasis—Emphysema of the eyelids—Blepharitis marginalis. **Other Diseases of the Eyelids**—Lagophthalmos—Blepharophimosis—Blepharospasm — Trichiasis and distichiasis — Symblepharon — Ankyloblepharon — Ptosis—Ectropion—Entropion. **Injuries of the Eyelids**—Wounds of the lids—Burns and scalds of the eyelids—Foreign bodies in the eyelids. **Operations on the Eyelids**—Chalazion operations—Operations for ptosis—Tarsorrhaphy—Canthoplasty—Trichiasis operations—Entropion operations—Epicanthus operations—Ectropion operations—Blepharoplastic operations—Blepharoplasty without a pedicle.

CHAPTER VI.

DISEASES OF THE LACRIMAL APPARATUS 219

Diseases of the Lacrimal Gland—Inflammation of the lacrimal gland—Dacryoadenalgia—Simple hypertrophy or adenoma of the lacrimal gland—Atrophy of the gland—Dacryoliths—Dacryops—Hydatid cyst—Fistula of the lacrimal gland—Dislocation of the gland—Syphilis of the lacrimal gland—Tumors of the lacrimal gland—Affections of the accessory glands. **Dis-**

PAGE

eases of the Drainage Apparatus—The puncta—The canaliculi—Leptothrix lacrimalis—Lacrimal actinomycosis—Inflammation of the lacrimal sac—Catarrhal dacryocystitis—Suppurative dacryocystitis—Obstruction and stricture of the lacrimo-nasal duct—Fistula of the lacrimal sac. **Lacrimal Operations.**

CHAPTER VII.

DISEASES OF THE CONJUNCTIVA 239

Congenital Anomalies. Tumors of the Conjunctiva—Dermoid tumor—Subconjunctival lipoma—Fibroma—Polypi, papillomata, and granulation tumors—Vascular tumors—Osteoma—Cysts—Lymphectasiæ—Sarcoma—Epithelioma. **Inflammations of the Conjunctiva**—Conjunctival injection—Hyperemia of the conjunctiva—Conjunctivitis—Simple conjunctivitis—Acute contagious conjunctivitis—Subacute catarrhal conjunctivitis—Chronic catarrhal conjunctivitis—Follicular conjunctivitis—Drug conjunctivitis—Vernal conjunctivitis—Purulent conjunctivitis—Gonorrheal ophthalmia of the adult—Ophthalmoblennorrhea neonatorum—Non-gonorrheal purulent conjunctivitis—Croupous conjunctivitis—Diphtheritic conjunctivitis—Phlyctenular conjunctivitis—Herpes of the conjunctiva—Papular conjunctivitis—Pemphigus of the conjunctiva—Parinaud's conjunctivitis—Lithiasis of the conjunctiva—Egyptian and military conjunctivitis—Variolar conjunctivitis—Chronic conjunctivitis—Lacrimal conjunctivitis—Granular conjunctivitis—Vaccinia of the conjunctiva—Varicella of the conjunctiva—Conjunctivitis petrificans. **Miscellaneous Diseases of the Conjunctiva**—Ophthalmia nodosa—Filaria—Pterygium—Pseudopterygium—Pinguecula—Amyloid degeneration of the conjunctiva—Xerosis—Tuberculosis of the conjunctiva—Argyrosis of the conjunctiva—Leprosy—Lupus of the conjunctiva—Hemorrhage from the conjunctiva—Abscess of the conjunctiva—Actinomycosis of the conjunctiva—Syphilis of the conjunctiva—Conjunctival ulcers—Subconjunctival ecchymosis—Emphysema of the conjunctiva—Chemosis—Symblepharon. **Injuries to the Conjunctiva**—Foreign bodies—Burns of the conjunctiva. **Affections of the Caruncle**—The plica semilunaris and caruncle—Tumors of the caruncle. **Operations on the Conjunctiva**—Pterygium operations—Symblepharon operations—Skin-grafting in symblepharon—Removal of tarsus and retrotarsal folds—Excision of the upper *cul-de-sac*—Expression of the contents of trachoma follicles—Grattage of the conjunctiva—Periectomy—Peritomy—Subconjunctival injections.

CHAPTER VIII.

DISEASES OF THE CORNEA 303

Congenital Anomalies—Opacities—Pigmentation—Congenital ectasia—Hydrophthalmos—Megalocornea and keratoglobus—Megalophthalmos. **Tumors of the Cornea. Classification of Corneal Inflammations. Suppurative Keratitis**—Ulcer of the cornea—Creeping ulcer of the cornea—Treatment of corneal ulcers—Neuroparalytic keratitis—Lagophthalmic keratitis—Xerophthalmic keratomalacia. **Non-suppurative Keratitis**—Phlyctenular keratitis—Interstitial keratitis—Vascular keratitis Keratitis bullosa—Aspergillar keratitis—Malarial keratitis Filamentary keratitis—Keratitis punctata—Superficial punctate keratitis—Ribbon-shaped corneal opacity—Herpes of the cornea—Marginal keratitis—Striped keratitis Disc-like keratitis—Grill-like keratitis. **Miscellaneous Diseases of the Cornea**—Discoloration of the cornea with blood-pigment—Sclerosis and atrophy of the limbus—Siderosis of the cornea—Xanthelasma of the cornea—Tuberculosis of the cornea—Lepra of the cornea. **Protrusions of the Cornea**—Staphyloma of

PAGE

the cornea — Keratoconus — Keratoglobus — Keratectasia — Fistula of the cornea. **Opacities of the Cornea**—Arcus senilis—Opacities due to keratitis. **Injuries of the Cornea**—Mechanical injuries of the cornea—Healing of corneal wounds—Hemorrhage into the cornea—Foreign bodies in the cornea. **Operations on the Cornea**—Abrasion of the cornea—Paracentesis of the cornea—Cauterization of the cornea—Excision of the cornea—Keratotomy—Operation for fistula of the cornea—Temporary renversement of a corneal flap—Tattooing of the cornea—Keratoplasty.

CHAPTER IX.

DISEASES OF THE SCLERA 361

Congenital Anomalies. Tumors of the Sclera. Inflammations of the Sclera—Episcleritis—Fugacious episcleritis—Scleritis. **Protrusions of the Sclera. Injuries of the Sclera.**

CHAPTER X.

DISEASES OF THE IRIS 374

Congenital Anomalies—Coloboma of the iris—Persistent pupillary membrane—Corectopia—Polycoria—Aniridia—Heterochromia—Exfoliation of the iris—Melanoma—Ectropion of the uvea—Albinism—Congenital tumors of the iris—Congenital atrophy of the iris—Adhesion of the iris to the cornea. **Tumors of the Iris**—Cysts of the iris—Sarcoma of the iris—Vascular tumors of the iris—Unusual tumors of the iris—Leprosy of the iris—Tubercle of the iris—Cysticercus—Filaria. **Inflammation of the Iris**—Iritis—Varieties of iritis—Uvéite. **Injuries of the Iris**—Iridodialysis—Anteversion of the iris—Laceration of the sphincter of the iris—Inversion of the iris—Traumatic irideremia—Prolapse of the iris—Foreign bodies. **Motor Disturbances of the Iris**—Mydriasis, miosis—Hippus—Iridodonesis. **Changes in the Anterior Chamber**—Depth—Contents. **Operations on the Iris**—Iridectomy—Transfixion of the iris—Iridenkleisis—Iridocapsulotomy—Iridocystectomy.

CHAPTER XI.

DISEASES OF THE CILIARY BODY 404

Coloboma of the ciliary body—Tumors of the ciliary body—Gumma of the ciliary body—Serous cyclitis—Plastic cyclitis—Purulent cyclitis—Injuries of the ciliary body.

CHAPTER XII.

DISEASES OF THE CHORIOID 411

Congenital Anomalies—Coloboma of the chorioid and retina—Albinism. **Tuberculosis of the Chorioid**—Tubercles of the chorioid—Large tubercular mass. **Tumors of the Chorioid**—Carcinoma of the chorioid—Other tumors of the chorioid—Sarcoma of the chorioid—Myoma of the chorioid—Ossification of the chorioid. **Inflammation of the Chorioid**—Hyperemia of the chorioid—Exudative chorioiditis—Purulent chorioiditis. **Injuries of the Chorioid**—Wounds of the chorioid—Foreign bodies—Chorioidal hemorrhage—Detachment of the chorioid—Rupture of the chorioid—Colloid disease of the macular region—Atrophy of the eyeball—Phthisis bulbi—Ophthalmomalacia.

CHAPTER XIII.

DISEASES OF THE CRYSTALLINE LENS 437

Congenital Anomalies—Coloboma of the lens—Microphakia—Lenticonus—Congenital aphakia—Congenital luxation of the lens. **Cataract**—Senile cataract—Congenital cataract—Soft cataract—Traumatic cataract—Secondary cataract—Complicated cataract—Diabetic cataract. **Operations for**

PAGE

Cataract—Preparation of the patient—Choice of an anesthetic—Extraction of senile cataract—Accidents during and following the operation—Peculiarities in the healing process—Choice of cataract extractions—Preliminary iridectomy—Should both eyes be operated on at the same sitting?—Other methods of extraction—Extraction of immature cataract—Scleronyxis—Operations for juvenile cataract—Operations for traumatic cataract—Procedures for post-operative cataract—Artificial ripening of cataract—Operations for artificial ripening—Refraction after extraction—Results of cataract operations—Spontaneous cure of cataract. **Dislocation of the Lens, etc.**—Acquired dislocation of the lens—Parasites in the lens—Spontaneous extrusion of the lens—Ossification of the lens—Regeneration of the lens—Injuries of the lens—Foreign bodies in the lens.

CHAPTER XIV.

DISEASES OF THE VITREOUS HUMOR 473

Coloboma of the vitreous humor—Persistent hyaloid artery—Inflammation of the vitreous humor—Suppurative hyalitis—Opacities of the vitreous humor—Hemorrhage into the vitreous humor—Fluidity of the vitreous—Synchysis scintillans—Animal parasites in the vitreous humor—Formation of new vessels in the vitreous—Pseudo-neuroepithelioma—Foreign bodies in the vitreous—Detachment of the vitreous—Retinitis proliferans.

CHAPTER XV.

DISEASES OF THE RETINA 481

Congenital Anomalies. Tumors of the Retina—Retinal cysts—Neuroepithelioma. **Parasites**—Subretinal cysticercus—Subretinal echinococcus. **Retinal Changes from Circulatory Disturbances**—Anemia of the retina—Ischemia of the retina—Hyperemia of the retina—Retinal vasculitis and perivasculitis—Aneurism of the retinal vessels—Apoplexy of the retina—Subhyaloid hemorrhage—Embolism of the central retinal artery—Thrombosis of the central retinal artery—Thrombosis of the central retinal vein. **Inflammation of the Retina**—Simple, or serous, retinitis—Syphilitic retinitis—Parenchymatous retinitis—Leucocythemic retinitis—Diabetic retinitis—Albuminuric retinitis—Gouty retinitis—Symmetrical macular changes in infancy—Retinitis circinata—Retinitis from exposure to excessive light—Punctate conditions of the fundus—Purulent retinitis—Purulent traumatic retinitis—Purulent secondary retinitis—Purulent metastatic, or embolic, retinitis—Septic retinitis. **Retinal Scleroses**—Retinitis proliferans—Striate retinitis—Pigment-streaks on the fundus—Pigmentary degeneration of the retina. **Injuries of the Retina**—Traumatic retinitis—Retinal hemorrhage from trauma—Traumatic detachment of the retina—Rupture of the retina—Pigmentation of the retina following trauma—Traumatic anesthesia of the retina—Traumatic perforations of the macula lutea. **Detachment of the Retina. Functional Diseases of the Retina**—Hyperesthesia of the retina—Anesthesia of the retina—Tuberculosis of the retina.

CHAPTER XVI.

DISEASES OF THE OPTIC NERVE 528

Congenital Anomalies—Absence of the optic nerve—Atrophy of the nerve—Coloboma of the optic nerve—Congenital cupping and other conditions of the disc. **Tumors**—Primary intradural tumors of the optic nerve—Primary extradural tumors of the optic nerve—Hyalin bodies in the nerve-head. **Inflammation of the Optic Nerve**—Intra-ocular optic neuritis—Retrobulbar optic neuritis—Optic neuritis with nasal discharge—Spurious optic

PAGE

neuritis. **Toxic Amblyopias**—Toxic amblyopia—Quinin-amaurosis—Methyl-alcohol amblyopia—Ptomain poisoning. **Atrophy of the Optic Nerve**—Primary optic-nerve atrophy—Secondary optic-nerve atrophy—Retinal type of optic-nerve atrophy—Embolic atrophy—Hereditary optic-nerve atrophy—Hemorrhage into the sheath of the optic nerve. **Disturbances of Vision without apparent Lesion**—Congenital amblyopia—Acute amaurosis following infantile convulsions—Congenital amblyopia for colors—Amblyopia ex anopsia—Uremic amaurosis—Diabetic amaurosis—Amblyopia from hemorrhage—Simulated amblyopia—Nyctalopia—Hemeralopia—Scotoma scintillans—Snow-blindness—Hemianopsia—Reflex amblyopia—Hysteric amblyopia—Colored vision—Malarial amblyopia—Micropsia, megalopsia, and metamorphopsia. **Injuries of the Optic Nerve. Operations on the Optic Nerve**—Opticociliary neurotomy—Opticociliary neurectomy—Value of these procedures.

CHAPTER XVII.

GLAUCOMA 562

Normal intra-ocular tension—Glaucoma: general considerations—Clinical varieties and treatment of primary glaucoma—Glaucoma simplex—Chronic irritative glaucoma—Acute inflammatory glaucoma—Hemorrhagic glaucoma—Glaucoma absolutum—Treatment of the apparently unaffected eye in glaucoma—Glaucoma after cataract operation—Prevention of primary glaucoma—Hydrophthalmos—Secondary glaucoma—Hypotony—Operations for glaucoma—Iridectomy—Iridectomy in the aphakic eye—Sympatheticectomy—Large paracentesis of the sclerotic with cyclicotomy—Sclerotomy.

CHAPTER XVIII.

SYMPATHETIC EYE DISEASES: INDICATIONS FOR ENUCLEATION 594

Sympathetic irritation—Sympathetic ophthalmitis—Indications for enucleation—Enucleation and its substitutes—Technique of evisceration—Evisceration with insertion of an artificial vitreous—Sclero-optic neurectomy—Implantation of a sphere into Tenon's capsule—Immunity to sympathetic ophthalmitis—Advantages of Mules's operation *versus* enucleation—Ocular prosthesis—Operation for the better support of an artificial eye—Operations for prosthesis in cicatricial orbit.

CHAPTER XIX.

DISEASES OF THE ORBIT 616

Congenital Anomalies—Cyclopia—The orbital contents—Anophthalmos—The ocular muscles—Congenital cysts of the orbit—Meningocele and encephalocele. **Tumors of the Orbit**—Etiology and location—Innocence or malignancy—Varieties of tumors—Lipoma—Angioma—Lymphangioma—Chondroma—Sarcoma—Osteoma—Plexiform neuroma—Epithelial tumors—Cysts of the orbit. **Inflammations of the Orbit**—Periostitis—Periostosis—Hyperostosis—Exostosis—Orbital cellulitis and phlegmon—Inflammation of the oculo-orbital fascia—Diseases of the frontal, ethmoidal, maxillary, and sphenoidal sinuses—Cavernous-sinus thrombosis—Hemorrhage into the orbit—Pulsating exophthalmos—Intermittent exophthalmos—Enophthalmos—Exophthalmic goitre. **Wounds and Injuries of the Orbit**—Contusion—Injuries to the orbital margin—Wounds of the soft tissues of the orbit—Luxation and avulsion of the globe—Fracture of the orbital bones—Foreign bodies in the orbit. **Operations on the Orbit**—Removal of tumors—Krönlein's operation—Exenteration of the orbit.

CHAPTER XX.

PAGE

ANOMALIES OF THE MUSCULAR APPARATUS 654

Action of the muscles—Binocular movements—General symptomatology—Special symptomatology—Cerebral lesions—Nuclear palsies—Ophthalmoplegia—Prognosis of ocular paralyses—Paralytic mydriasis—Paralytic miosis—Paralysis of accommodation—Spastic strabismus—Nystagmus—Deviation of the visual axes of non-paralytic origin—Determination of the angle of squint—Latent deviation—Operations on the ocular muscles.

CHAPTER XXI.

ERRORS OF REFRACTION 686

Properties of Optical Lenses—Light—Refraction—Convex and concave lenses—Numbering of lenses. **Emmetropia. Ametropia. Hypermetropia**—Definition — Etiology — Pathology — Symptoms — Diagnosis — Treatment. **Myopia** — Definition — Etiology — Pathology — Symptoms — Diagnosis — Treatment. **Astigmatism** — Definition — Varieties — Etiology — Symptoms —Treatment. **Aphakia** — Definition — Etiology — Symptoms — Diagnosis — Treatment. **Presbyopia** — Definition — Etiology — Diagnosis — Treatment. **Antimetropia and Anisometropia**—Definition—Etiology—Diagnosis—Treatment.

CHAPTER XXII.

THE OCULAR MANIFESTATIONS OF NERVOUS DISEASES 733

General Considerations—Conjugate lateral paralysis—Ptosis—Various paralyses—Nystagmus. **Conditions of the Pupil**—Miosis—Mydriasis. **The Visual Tract. The Field of Vision**—Hemianopsia. **Focal Lesions of the Brain**—Optic neuritis—Hemianopsia—Hemianoptic pupil-symptom of Wernicke—Visual amnesia—Alexia—Visual aphasia—Dyslexia—Amnesic color-blindness—Visual hallucinations. **Ocular Symptoms of Organic Diseases of the Brain and Spinal Cord**—Ocular symptoms of meningitis—Multiple sclerosis—Locomotor ataxia—Pseudobulbar paralysis of cerebral origin—Combined sclerosis of the spinal cord—Friedreich's ataxia—Acute ascending paralysis—Myelitis—Multiple neuritis—Migraine—Neuralgia—Herpes zoster—General paralysis of the insane—Insanity—Paralysis agitans—Infantile paralysis—Tetany—Hydrocephalus—Acromegalia—Thomsen's disease—Syringomyelia and Morvan's disease—Epilepsy—Chorea—Exophthalmic goitre. **Asthenopia, Amblyopia, and Amaurosis of Nervous Origin**—Neurasthenia — Hysteria — Neurosis following traumatism. **Other Forms of Amblyopia and Amaurosis** — Amblyopia caused by a disturbance of the cerebral circulation—Reflex amblyopia—Congenital amblyopia—Pretended amblyopia and amaurosis—Tests for binocular blindness—Tests for non-ocular amblyopia—Erythropsia.

CHAPTER XXIII.

PREPARATION FOR OPHTHALMIC OPERATIONS 749

Qualifications of the surgeon—Preparation of the surgeon and assistants—Preliminary preparation of the patient—Time for operation—Preparation of the region of operation—Assistants—Preparation of the instruments—Preparation of mops and dressings—Local hemostasis—The operation room.

CHAPTER XXIV.

THE HYGIENE OF THE EYES 756

Care of the Eyes at Birth, during Infancy, and in early Childhood—Kindergartens—Light—Print—Reading. **Care of the Eyes during School-**

PAGE

life—Construction of school-buildings in relation to hygiene—Prevalence of myopia in the public schools. **The Influence of Injurious Habits on the Eyes of Adults**—The effect of occupation upon the eyes—Care of the eyes during epidemics. **Care of the Eyes in Old Age**—Climatic changes in their effects upon the eyes—Care of spectacles.

CHAPTER XXV.

METHODS EMPLOYED IN THE MICROSCOPIC EXAMINATION OF THE EYE..... 772

Material—Preservation of eyeballs for demonstration—Fixing—Washing—Decalcification—Bleaching—Hardening—Cutting the eyeball preparatory to mounting—Imbedding—Cutting the sections—Staining—Nuclear stains—Protoplasmic stains—Dehydration of the sections—Clearing of the sections—Mounting the sections on slides—Demonstration of definite substances and tissue-elements—Demonstration of foreign substances in the eye—Staining for bacteria in sections.

INDEX 793

LIST OF ILLUSTRATIONS.

FIG.		PAGE
1.	Transverse section through the brain of a chick: three days' incubation. (AUTHOR.)	1
2.	The secondary eye-vesicle, with lens-fossette. (After VON KÖLLIKER.)	2
3.	Constriction of the lens. (After VAN BAMBEKE.)	2
4.	The eye-stalk and the eye. (KOLLMANN.)	3
5.	Tunica vasculosa lentis. (After O. SCHULTZE.)	4
6.	Scheme of the secondary eye-vesicle. (MANZ-ZIEGLER.)	5
7.	Vertical section of the anterior half of an eye from a human embryo of eight or nine weeks. (After VON KÖLLIKER.)	5
8.	Section of two ciliary processes of a human embryo, enlarged 200 diameters. (After KRISCHEWSKI.)	6
9.	Eye of a human embryo of seven weeks, enlarged 20 diameters. (KOLLMANN.)	6
10.	Inner wall of the orbit and adjacent parts. (HIS.)	8
11.	Frontal section showing the relation of the orbits to the other cavities of the skull. (After ZUCKERKANDL.)	10
12.	Capsule of Tenon. (MERKEL.)	11
13.	Horizontal section of the right orbit, viewed from above. (After GERLACH.)	12
14.	Position of eyeball in the right orbit and the location of the orbital muscles. (MERKEL.)	13
15.	The right orbit opened from behind. (AUTHOR.)	14
16.	Sagittal section of the orbit. (After DEAVER.)	15
17.	Dissection of base of left orbit. (AUTHOR.)	16
18.	Distribution of the third and sixth nerves in the orbit. (LÉVEILLÉ.)	16
19.	Ophthalmic division of the fifth nerve. (LÉVEILLÉ.)	17
20.	Lateral view of the orbit, showing the nerves. (After DEAVER.)	18
21.	The ophthalmic artery and its branches, viewed from above. (After MERKEL and KALLIUS.)	20
22.	Distribution of the ophthalmic vein. (GURWITSCH.)	21
23.	The nasal duct and lacrimal gland. (AUTHOR.)	23
24.	Probes introduced into the canaliculi to show their direction. (AUTHOR.)	24
25.	Section of the upper eyelid. (AUTHOR.)	25
26.	Arteries of the (right) eyelids. (AUTHOR.)	27
27.	Veins of the (left) eyelids. (After SOEMMERING.)	28
28.	Antero-posterior section of the eyeball. (LÉVEILLÉ.)	29
29.	Section through the human cornea. (BÖHM and DAVIDOFF.)	30
30.	Corneal corpuscles of the dog. (BÖHM and DAVIDOFF.)	31
31.	The iridocorneal angle. (AUTHOR.)	32
32.	Corneal nerves of the pig. (After ROLLETT.)	33
33.	Radial section of the human iris. (AUTHOR.)	34
34.	Diagram of the vessels of the eye. (LEBER.)	35
35.	Injected blood-vessels of the iris, ciliary body, and chorioid of man. × 7. BÖHM and DAVIDOFF.)	36
36.	Meridional section of the human ciliary body. (After BÖHM and DAVIDOFF.)	37
37.	Meridional section of human ciliary processes. (PIERSOL.)	38
38.	Section through the ciliary region of an hypermetropic eye. (IWANOFF.)	39
39.	Section through the ciliary region of a myopic eye. (IWANOFF.)	39
40.	Bleached section showing glands of the ciliary body. × 300. (TREACHER COLLINS.)	40
41.	Vertical section of the chorioid. (BÖHM and DAVIDOFF.)	40
42.	Diagram of occipital region of right cerebral hemispheres	42
43.	The macula near the fovea centralis. × 80	43
44.	Hexagonal cells from the pigment layer of the retina of a rabbit. (AUTHOR.)	44
45.	Diagram of the structure of the human retina according to Golgi's method. (GREEFF.)	45
46.	Photomicrograph of the optic chiasma, showing decussation of the nerve-fibres. (AUTHOR.)	48
47.	Transverse section of orbital part of the optic nerve behind the entrance of the central vessels. (After MERKEL.)	49
48.	Transverse section of the orbital part of the optic nerve in front of the entrance of the central vessels. (After MERKEL.)	50
49.	Isolated lens-fibres. (After J. ARNOLD.)	52
50.	Transverse section of lens-fibres. (After J. ARNOLD.)	52
51.	Anterior surface of the lens of an adult. (After J. ARNOLD.)	53
52.	Marginal whorl of the lens, showing the transition of the epithelium into lens-fibres. (SCHULTZE.)	53
53.	Epithelium of the inner surface of the anterior capsule. (SCHULTZE.)	54
54.	Dissection to show the zonula. (After SCHULTZE.)	55
55.	Diagram of lymph-spaces of the eyeball. (After FUCHS.)	56
56.	The normal fundus of the right eye. (HENLE.)	57
57.	Diagram of the retinal vessels of the left eye	58
58.	Injected blood-vessels of the macular region. (BÖHM and DAVIDOFF.)	59
59.	Diagram of a simple optical system	62
60.	Action of a biconvex lens upon rays of light	63
61.	Action of a convex lens on rays of light proceeding from a point within the focus	63
62.	The image formed by a biconvex lens	64

FIG.		PAGE
63.	Diagram of the reduced eye	65
64.	Diagram to show that the visual angle and size of the retinal image vary with the distance of the object from the eye	66
65.	Purkinje-Sanson images	67
66.	Diagram showing the corneal axis, the optic axis, the visual line, the line of fixation, and the three angles	68
67.	Diagram to show spheric aberration	69
68.	Diagram to show chromatic aberration	69
69.	Diagram to show the primary position of the right eye, the eye turned upward and inward, and downward and outward	74
70.	The horopteric circle of Joannes Müller	76
71.	Diagram of the field of binocular vision	77
72.	Diagram of the normal visual field for white and colors. (JENNINGS.)	78
73.	Diagram to show the blind spot in the visual field	79
74.	Diagram to show irradiation	81
75.	Diagram of the decomposition of solar light into the spectral colors. (JENNINGS.)	82
76.	Eversion of the upper eyelid	88
77.	Examination of the eye of a child	89
78.	Noyes's retractor	90
79.	Position for secondary eversion of the upper lid	90
80.	Diagram to show the vascular systems of the eye	91
81.	Diagram to show focal (or oblique) illumination	93
82.	Placido's disc	94
83.	Jackson's binocular magnifier	95
84.	Opacities in the lens	99
85.	Method of taking the tension of the eye	100
86.	The Maddox rod	102
87.	Diagram to show the Maddox rod test for horizontal deviations	103
88.	Diagram to show the Maddox rod test for vertical deviations	103
89.	The von Graefe test, used at the reading distance	104
90.	The Maddox double prism as a near test	104
91.	Use of the Maddox double prism for far	105
92.	Diagram to show use of the Maddox double prism for near	106
93.	Diagram to show the use of the Maddox double prism in testing the oblique muscles	107
94.	Stevens's improved rotating prism slide	107
95.	Stevens's phorometer ready for use	108
96.	Risley's rotary prism	108
97.	Prism-holder. (NOYES.)	109
98.	Prism-holder. (HULEN.)	110
99.	Tropometer. (STEVENS.)	111
100.	Scale of the tropometer	112
101.	Measurement of strabismus by the perimeter	113
102.	Measurement of convergent strabismus of right eye by Priestley Smith's method	114
103.	Strabismometer	116
104.	Linear measurement of strabismus	116
105.	Schematic section of the Kagenaar model of the Javal-Schioetz ophthalmometer. (KAGENAAR.)	117
106.	Appearance of the mires in the primary position. (KAGENAAR.)	118
107.	Appearance of the mires in the secondary position, showing four dioptres of astigmatism. (KAGENAAR.)	118
108.	The visual angle	119
109.	Snellen's test-type	119
110.	Wallace's test-type	119
111.	Improved trial-frame	120
112.	The hair-optometer	122
113.	Measurement of the near point	123
114.	The McHardy perimeter	125
115.	Förster's photometer. (FICK.)	128
116.	Wools for the detection of color-blindness. (OLIVER.)	130
117.	Color-sense measure. (OLIVER.)	131
118.	Morton's ophthalmoscope	135
119.	Loring's ophthalmoscope	136
120.	Direct ophthalmoscopy	137
121.	Indirect ophthalmoscopy	137
122.	Relative positions in direct ophthalmoscopy	138
123.	Indirect ophthalmoscopy	139
124.	Localization of opacities in the lens and cornea. (FICK.)	140
125.	Ophthalmoscopy in emmetropia	142
126.	Measurement of hypermetropia by direct ophthalmoscopy	142
127.	Measurement of hypometropia (myopia) by direct ophthalmoscopy	143
128.	Diagram to show parallactic displacement in indirect ophthalmoscopy	144
129.	Diagram to explain retinoscopy with the concave mirror. (After LANDOLT.)	145
130.	Retinoscopy mirror	147
131.	Thorington's light-screen with iris-diaphragm	148
132.	Movement of the shadow in myopia	148
133.	Movement of the shadow in emmetropia, low myopia, and hypermetropia	149
134.	Movement of the shadow in mixed astigmatism	150
135.	Diagram to show correction of astigmatism	151
136.	Thorington's axonometer	151
137.	Test for simulated blindness. (FRIDENBERG.)	153
138.	Apparatus for fixing the head in x-ray examination. (DAVIDSON.)	155
139.	The crossthread localizer. (DAVIDSON.)	156
140.	Herpes zoster ophthalmicus. (FUCHS.)	161
141.	Blastomycetic dermatitis involving the eyelids. (WALKER.)	166
142.	Dermoid tumor. (AUTHOR.)	168
143.	Verruca of the eyelids. (VON AMMON.)	169
144.	Cornu cutaneum. (SCHAW.)	169
145.	Solid edema of the eyelids. (CRITCHETT.)	170

FIG.		PAGE
146.	Elephantiasis of the upper eyelid. (After WALZBERG.)	171
147.	Varicose vein of the upper eyelid. (AUTHOR.)	171
148.	Gummata of the eyelids. (KEBER.)	174
149.	Microscopic section of an epithelioma of the eyelid. (AUTHOR.)	176
150.	Rodent ulcer. (GRINDON.)	178
151.	Microscopic section of a chalazion. (AUTHOR.)	181
152.	Varioloid of the eyelids. (VON AMMON.)	183
153.	Cicatricial ectropion. (AUTHOR.)	192
154.	Burn of face and eyelids. (VAIL.)	194
155.	Result of burn of face and eyelids. (VAIL.)	195
156.	Entropion forceps. (AUTHOR.)	196
157.	Wilder's operation for ptosis	197
158.	Incisions and sutures in Panas's operation	198
159.	Appearance of the eye after Panas's operation	199
160.	Operation for ptosis. (TANSLEY.)	199
161.	Tarsorrhaphy by von Graefe's method. (CZERMAK.)	200
162.	Tarsorrhaphy by Fuchs's method. (CZERMAK.)	200
163.	Von Ammon's canthoplasty. (After CZERMAK.)	201
164.	The sutures in place in canthoplasty. (After CZERMAK.)	201
165.	Excision operation for trichiasis. (After STEPHENSON.)	202
166.	Jaesche-Arlt operation for trichiasis	203
167.	Section of the upper eyelid in Snellen's operation for entropion	204
168.	The method of placing the sutures in Snellen's operation for entropion	204
169.	Hotz's operation for entropion	205
170.	Harlan's operation for entropion	205
171.	Von Ammon's operation for epicanthus	206
172.	Wicherkiewicz's operation for epicanthus	207
173.	Snellen's thread operation for ectropion	207
174.	Von Langenbeck's operation for ectropion	208
175.	Von Ammon's operation for ectropion	209
176.	Dieffenbach's operation for ectropion	209
177.	Sanson's operation for ectropion	209
178.	Robertson's first operation for ectropion	210
179.	Robertson's latest operation for ectropion	210
180.	Kuhnt's operation for ectropion	211
181.	Supporting stitch in Kuhnt's operation	211
182.	Müller's ectropion operation	212
183.	Hotz's operation for cicatricial ectropion of the lower eyelid	212
184.	Hotz's operation for cicatricial ectropion of the upper eyelid	213
185.	Fricke's blepharoplasty	213
186.	Blasius's blepharoplasty	214
187.	Blasius's blepharoplasty	214
188.	Hasner d'Artha's blepharoplasty for both lids	215
189.	Dieffenbach's blepharoplasty	215
190.	The Dieffenbach-Szymanowski operation	215
191.	Knapp's blepharoplasty	216
192.	Outlines of incision in blepharoplasty. (POSEY and SHUMWAY.)	216
193.	Result of blepharoplasty. (POSEY and SHUMWAY.)	217
194.	Probes passed into the canaliculi to show their direction. (AUTHOR.)	232
195.	Lacrimal syringe. (MCFARLAN.)	233
196.	Apparatus for lavage of the naso-lacrimal duct. (AUTHOR.)	234
197.	Lacrimal probe. (SNELLEN.)	235
198.	Lacrimal probe. (CALDWELL.)	237
199.	Dermoid tumor of the conjunctiva. (VON AMMON.)	240
200.	Microscopic section of a conjunctival dermoid tumor. (AUTHOR.)	241
201.	Polypi of the conjunctiva. (DE BECK.)	242
202.	Subconjunctival cyst. (AUTHOR.)	243
203.	Microscopic section of an epibulbar leucosarcoma with hematogenous pigmentation. (KERSCHBAUMER.)	244
204.	Antero-posterior section of primary sarcoma of the conjunctiva. (AUTHOR.)	244
205.	Growth of conjunctival epithelioma into the cornea. (DE BECK.)	245
206.	Microscopic section of an epithelioma of the conjunctiva advancing into the cornea. (DE BECK.)	245
207.	Eye-drop bottle (CHALK) and aseptic flask (STROSCHEIN)	247
208.	Congestion of the posterior conjunctival vessels. (AUTHOR.)	248
209.	Ciliary injection. (AUTHOR.)	249
210.	Section of growth in the bulbar type of vernal conjunctivitis. (DANVERS.)	262
211.	Trachoma follicles. (AUTHOR.)	279
212.	Conjunctivitis petrificans. (REIF.)	285
213.	Traumatic pterygium. (OLIVER.)	286
214.	Tuberculosis of the conjunctiva. (EYRE.)	290
215.	Eye-speculum. (AUTHOR.)	296
216.	Operation for pterygium. (MCREYNOLDS.)	298
217.	Operation for symblepharon. (TEALE.)	298
218.	Operation for symblepharon. (HARLAN.)	299
219.	Excision of the upper cul-de-sac. (After STEPHENSON.)	300
220.	Congenital ectasia of the cornea. (WÜRDEMANN.)	304
221.	Appearance of the cornea in ulcus serpens. (KNIES.)	312
222.	Creeping ulcer of the cornea. (AUTHOR.)	313
223.	Keratitis e lagophthalmo. (AUTHOR.)	318
224.	Section of the cornea in phlyctenular keratitis. (After IWANOFF.)	321
225.	Section of the cornea in interstitial keratitis. (BAAS.)	322
226.	Notched teeth. (DE BECK.)	323
227.	Pegged teeth. (DE BECK.)	323
228.	Trachoma with pannus. (AUTHOR.)	326
229.	Section of the cornea in pannus. (AUTHOR.)	327
230.	Microscopic section of growth in aspergillar keratitis. (AUTHOR.)	330
231.	Malarial keratitis. (KIPP.)	331

FIG.		PAGE
232.	Filamentary keratitis. (HESS.)	332
233.	Keratitis punctata. (AUTHOR.)	333
234.	Appearance of the cornea in punctate keratitis. (KNIES.)	334
235.	Appearance of the cornea in ribbon-shaped opacity. (KNIES.)	335
236.	Appearance of the cornea in herpes febrilis. (KNIES.)	337
237.	Section of the cornea in striate keratitis. (HESS.)	338
238.	Sclerosis and atrophy of the corneal limbus. (FUCHS.)	340
239.	Staphyloma of the cornea. (AUTHOR.)	342
240.	Keratoconus. (AUTHOR.)	344
241.	Keratoglobus. (After DEMOURS.)	345
242.	Fistulous ulcer of the cornea. (WÜRDEMANN.)	347
243.	True corneal fistula, showing downgrowth of epithelium. (AUTHOR.)	348
244.	Hemorrhage into the cornea. (After DE BECK.)	354
245.	The same eye one day later. (After DE BECK.)	354
246.	Operation for corneal staphyloma. (KNAPP.)	356
247.	Tattooing instrument. (BARCK.)	359
248.	Deep scleritis. (AUTHOR.)	363
249.	Sclerosing keratitis. (After DEMOURS.)	365
250.	Equatorial ectasiæ of the sclera. (After VON AMMON.)	367
251.	Cirsoid ciliary staphyloma. (VOSSIUS.)	367
252.	Posterior staphyloma of Scarpa	368
253.	Ciliary staphyloma. (PAGENSTECHER and GENTH.)	368
254.	Stitch for scleral rupture. (After NUEL.)	370
255.	Giant magnet	372
256.	Congenital coloboma of the iris. (SEGGEL.)	374
257.	Persistent pupillary fibres, forming loops. (DE BECK.)	375
258.	Forms of persistent pupillary membrane. (DE BECK.)	375
259.	Forms of corectopia. (DE BECK.)	375
260.	Polycoria. (DE BECK.)	376
261.	Cyst of the iris. (COLLINS.)	377
262.	Serous cyst of the iris following discission for milky cataract. (AYRES.)	378
263.	Cysticercus which has broken through an iridal vessel. (KRAEMER.)	380
264.	Pericorneal injection in iritis. (AUTHOR.)	382
265.	Different appearances of the pupil in iritis, after the use of atropin	383
266.	Gummatous iritis. (AUTHOR.)	385
267.	Iritis with hypopyon and chemosis. (VON AMMON.)	386
268.	Antero-posterior section of an eye which shows post-iritic glaucoma, calcareous cataract, and chorioiditis. (AUTHOR.)	387
269.	Proliferation of the pigment layer of the iris in diabetes. (AUTHOR.)	389
270.	Iridodialysis. (DE BECK.)	394
271.	First step in the operation of iridectomy. (AUTHOR.)	397
272.	Second step in the operation of iridectomy. (AUTHOR.)	397
273.	Third step in the operation of iridectomy. (AUTHOR.)	398
274.	Iridectomy in threatened corneal perforation	400
275.	Instruments used in operations on the iris. (AUTHOR.)	401
276.	Iridocapsulotomy. (DE WECKER.)	402
277.	Iridocapsulotomy. (DE WECKER.)	402
278.	Coloboma of the iris and ciliary body. (BOCK.)	404
279.	Leucosarcoma of the ciliary body and melanosarcoma of the iris. (KERSCHBAUMER.)	405
280.	Gumma of the ciliary body. (NORRIS and OLIVER.)	406
281.	Anterior part of eyeball with iridocyclitis. (After POLLOCK.)	408
282.	Coloboma of the chorioid. (BENSON.)	411
283.	Coloboma of the macula, with islets of pigment and an atrophic crescent of the optic disc. (SILEX.)	412
284.	Metastatic carcinoma of the chorioid	414
285.	Fungating sarcoma of the chorioid. (AUTHOR.)	415
286.	Early stage of chorioidoretinitis. (LIEBREICH.)	422
287.	Late stage of chorioidoretinitis. (LIEBREICH.)	423
288.	Senile areolar atrophy of the chorioid. (NETTLESHIP.)	424
289.	Types of posterior staphyloma. (GALEZOWSKI.)	425
290.	Panophthalmitis and orbital cellulitis. (W. T. SHOEMAKER.)	431
291.	Section of a panophthalmitic eye. (WÜRDEMANN.)	432
292.	Ruptures of the chorioid. (KNAPP.)	434
293.	Colloid change in the macular region. (DE SCHWEINITZ.)	435
294.	Binocular coloboma of the lens without coloboma of the iris or chorioid. (AUTHOR.)	437
295.	Lenticonus anterior. (Modified from WEBSTER.)	438
296.	Lenticonus posterior in a buphthalmic eye. (Modified from PERGENS.)	438
297.	Congenital luxation of the lens. (DE BECK.)	439
298.	Location of opacity in different forms of cataract	440
299.	Antérior capsular cataract of stellate form. (VON AMMON.)	440
300.	Appearance of cataract	441
301.	Shadow of iris seen in unripe cataract. (FUCHS.)	442
302.	Coralliform cataract. (AUTHOR.)	444
303.	Anterior polar cataract. (VON AMMON.)	445
304.	Calcareous cataract. (VON AMMON.)	446
305.	Anterior segment of a rabbit's eye twenty-four hours after extraction of the lens. (BATES.)	447
306.	Instruments for extraction of cataract without iridectomy. (AUTHOR.)	450
307.	Incision in cataract extraction. (AUTHOR.)	451
308.	Incision of the cornea in various methods of cataract extraction. (NIMIER and DESPAGNET.)	454
309.	Cystoid cicatrix lined by atrophied iris-tissue, in an eye in which glaucoma followed cataract extraction. (TREACHER COLLINS.)	457
310.	Depression of cataract. (AGATZ.)	463
311.	Discission with two needles	464
312.	Site of incision for traumatic cataract. (AUTHOR.)	465
313.	Operation for post-operative cataract. (KUHNT.)	466
314.	Dislocation of the lens into the anterior chamber. (VON AMMON.)	470

FIG.		PAGE
315.	Coloboma of the vitreous humor. (ECKER.)	473
316.	Canal of Cloquet, with cyst in the vitreous humor. (KOLLER.)	474
317.	Extensive vascular growth in the vitreous humor. (HARLAN.)	479
318.	Rosette of neuroepithelial cells with short protoplasmic processes. (WINTERSTEINER.)	483
319.	Tubulose structure of neuroepithelioma of the retina. (WINTERSTEINER.)	483
320.	Neuroepithelioma of the retina. (WINTERSTEINER.)	484
321.	Neuroepithelioma of the retina. (AUTHOR.)	485
322.	Pseudo-neuroepithelioma of the retina. (AUTHOR.)	487
323.	Perivasculitis of the retinal vessels. (HARLAN.)	491
324.	Arterio-venous aneurism of the retina. (FUCHS.)	492
325.	Subhyaloid hemorrhage. (HOTZ.)	494
326.	Section of the retina in embolism of the central artery. (After MARPLE.)	495
327.	Section through the optic nerve showing an embolus in the central artery. (After MARPLE.)	496
328.	Serous retinitis. (OLIVER.)	500
329.	Macular change in infancy. (TAY.)	512
330.	Proliferating retinitis. (JAEGER.)	518
331.	Coloboma of the optic nerve and chorioid. (POSEY.)	529
332.	Microscopic section showing hyalin bodies in the nerve-head. (AUTHOR.)	532
333.	Hyalin bodies in the optic disc. (NIEDEN.)	533
334.	Photomicrograph of the optic-nerve head in case of tumor of the brain. (AUTHOR.)	537
335.	Fields of vision in a case of quinin-amaurosis. (AUTHOR.)	543
336.	Deformity of the skull producing exophthalmos, divergent strabismus, and atrophy of the optic nerve. (RISLEY.)	547
337.	Atrophy of the optic nerve following phthisis bulbi. (AUTHOR.)	548
338.	Metamorphopsia varians. (DUDLEY.)	560
339.	Section of normal superior cervical ganglion. (AUTHOR.)	564
340.	Angle of the anterior chamber of a normal eye. (AUTHOR.)	565
341.	Angle of the anterior chamber of a glaucomatous eye. (AUTHOR.)	566
342.	Head of normal optic nerve. (AUTHOR.)	570
343.	Head of optic nerve in glaucoma absolutum. (AUTHOR.)	570
344.	Types of excavation of the optic nerve. (JENNINGS.)	571
345.	Field of vision of left eye in a case of glaucoma simplex. (AUTHOR.)	574
346.	Section of superior cervical ganglion in chronic irritative glaucoma. (AUTHOR.)	575
347.	Section of superior cervical ganglion of the sympathetic nerve in a case of hemorrhagic glaucoma. (AUTHOR.)	579
348.	Acquired hydrophthalmos. (AUTHOR.)	581
349.	Binocular hydrophthalmos following ophthalmia neonatorum. (AUTHOR.)	583
350.	Appearance of pupil after iridectomy for glaucoma	586
351.	Site of the incision for removal of the superior cervical ganglion of the sympathetic nerve. (AUTHOR.)	587
352.	Operation for excision of the superior cervical ganglion of the sympathetic nerve. (AUTHOR.)	588
353.	Posterior sclerotomy. (After CZERMAK.)	592
354.	Enucleation scissors	608
355.	Artificial-vitreous introducer. (TODD.)	611
356.	Sclero-optic neurectomy	612
357.	Sphere in position. (FOX.)	614
358.	Conformer. (FOX.)	614
359.	Cyclops. (After VAN DUYSE.)	616
360.	Anophthalmos. (AUTHOR.)	617
361.	Fibrosarcoma of the orbit. (WILSON.)	622
362.	Orbital tumor in a case of sarcomatosis. (W. T. SHOEMAKER.)	623
363.	Abscess of the orbit in a child ten days old, injured during instrumental delivery. (AUTHOR.)	631
364.	Thrombosis of the cavernous sinus. (W. T. SHOEMAKER.)	637
365.	Avulsion of the eyeball. (BARCK.)	647
366.	Krönlein's operation	652
367.	Modification of Krönlein's operation. (PARINAUD and ROCHE.)	653
368.	Scheme of the axes of rotation of the ocular muscles	654
369.	Muscles associated in moving the eyeballs in the directions indicated by the arrows	655
370.	Illustrating the projection of the false image (colored)	658
371.	Paralysis of the internal rectus of the right eye, producing crossed diplopia (colored)	659
372.	Paralysis of the external rectus of the right eye, producing homonymous diplopia (colored)	660
373.	Position of the images in ocular paralyses (colored)	661
374.	Mnemonic scheme of ocular paralyses. (WERNER.)	665
375.	Ophthalmoplegia. (AUTHOR.)	666
376.	Ophthalmoplegia. (AUTHOR.)	667
377.	Measurement of the angle of squint by the perimeter	672
378.	Linear measurement of squint	673
379.	Relative positions of the line of light and the candle-flame in the Maddox rod test (colored)	675
380.	Insufficiency of the oblique muscles	678
381.	Tenotomy of the external rectus muscle. (AUTHOR.)	682
382.	Advancement of an ocular muscle, showing the suture in position before tying	683
383.	Total advancement. (AUTHOR.)	684
384.	Capsular advancement. (FOX.)	685
385.	Passage of an oblique ray from a rare to a dense medium	686
386.	Refraction by plane glass	687
387.	Refraction by a prism	687
388.	Refraction by prisms	688
389.	Profiles of lenses	689
390.	Emmetropic eye	691
391.	Refraction of parallel rays of light in emmetropia, hypermetropia, and myopia	693
392.	Inability to focus parallel rays of light on the retina of the hypermetropic eye	694
393.	Accommodation is necessary to bring parallel rays on the retina of the hypermetropic eye	694

FIG.		PAGE
394.	Additional accommodation power is necessary to bring an object at the near point upon the retina of an hypermetropic eye	695
395.	Illustrating the focus of parallel rays of light in the myopic eye	703
396.	Only divergent rays are brought to a focus on the retina	704
397.	Parallel rays focused in the vitreous	704
398.	Chart showing increase of myopia in school-children of Philadelphia. (RISLEY.)	707
399.	Chart showing increase of myopia in school-children of St. Petersburg. (ERISMANN.)	707
400.	Chart showing disease in relation to refraction	708
401.	Chart showing vision and pain in relation to refraction	709
402.	Chart showing disease in relation to age	709
403.	The cornea in emmetropia	717
404.	Irregular astigmatism	717
405.	Regular astigmatism	717
406.	Regular astigmatism	717
407.	Refraction of rays in regular astigmatism	718
408.	Simple hypermetropic astigmatism	719
409.	Simple myopic astigmatism	719
410.	Compound hypermetropic astigmatism	720
411.	Compound myopic astigmatism	720
412.	Mixed astigmatism	721
413.	"Clock-dial" chart	722
414.	Range of accommodation at various ages. (DONDERS.)	727
415.	Diagram of the visual tract (colored)	735
416.	Diagram of right homonymous hemianopsia and of the sites of lesions which may cause it	737
417.	Diagram of Wernicke's pupil symptom	738

LIST OF FULL-PAGE PLATES.

PLATE		FACING PAGE
I.	Coronal Section, showing the Orbits and Adjacent Parts. (MURPHY.)	12
II.	Normal Fundus Oculi	54
III.	Normal Fundus Oculi	56
IV.	Normal Fundus Oculi	58
V.	Normal Fundus Oculi: Macular and Foveal Reflexes. (JOHNSON.)	60
VI.	Test-words for the Determination of Accommodation. (OLIVER.)	122
VII.	External Diseases of the Eye	180
VIII.	External Diseases of the Eye	226
IX.	Bacteria found in Conjunctival Inflammations. (SCHANZ.)	250
X.	External Diseases of the Eye	266
XI.	External Diseases of the Eye	308
XII.	External Diseases of the Eye	384
XIII.	Diseases of the Chorioid	416
XIV.	Diseases of the Chorioid	426
XV.	Diseases of the Retina. (OLIVER.)	488
XVI.	Diseases of the Retina	494
XVII.	Diseases of the Retina	498
XVIII.	Diseases of the Retina	504
XIX.	Diseases of the Retina	512
XX.	Diseases of the Retina	516
XXI.	Diseases of the Retina	522
XXII.	Diseases of the Optic Nerve	544
XXIII.	Diseases of the Optic Nerve: Glaucoma	562

MODERN OPHTHALMOLOGY.

CHAPTER I.

DEVELOPMENT OF THE EYE.

THE formation of the eye is initiated by a protrusion of the lateral walls of the primary cerebral vesicle. Thus the primary optic vesicles are formed, which detach themselves more and more from the brain until finally

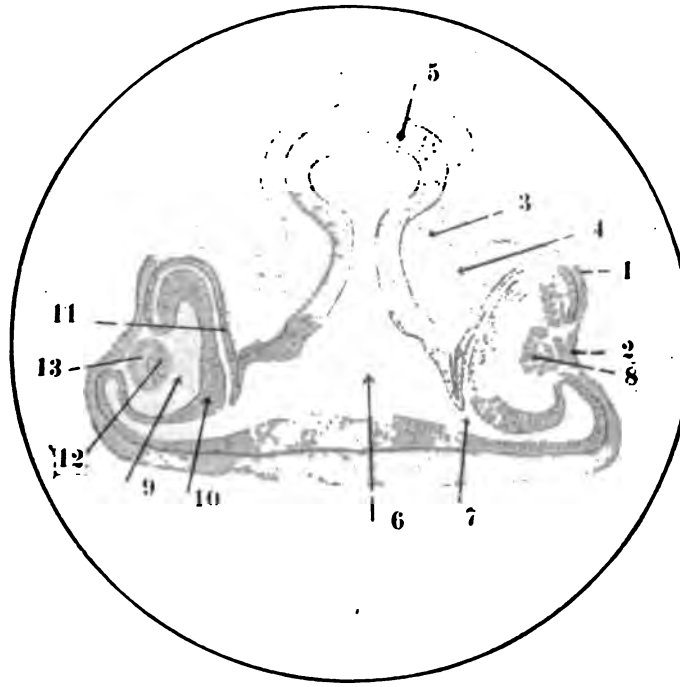


Fig. 1.—Transverse section through the brain of a chick : three days' incubation. (AUTHOR.)

(Original drawing by DR. CARL FISCH.)

1, Ectoderm. *2*, Cornea. *3*, Mesenchyma. *4*, Primitive artery. *5*, Wall of brain-vesicle. *6*, Cavity of brain-vesicle. *7*, Eye-stalk. *8*, Lens. *9*, Cavity of eye-vesicle. *10*, Anterior part of the retina. *11*, Posterior layer of the retina. *12*, Formation of lens-fibres. *13*, Cavity of lens-vesicle.

they are connected with it only by a slender peduncle, the eye-stalk. The vesicles are hollow, and their cavity communicates with the brain-cavity by means of the canal in the eye-stalk. The original form of the optic

vesicle soon undergoes a change by the appearance of two depressions on its lateral and inferior surfaces. The one leads to the formation of the lens, the other to that of the vitreous body.

The formation of the lens begins in the human fetus at the commencement of the fourth week. Where the ectoderm lies over the primary optic vesicle, a slight thickening is observed, which soon deepens into a slight

Fig. 2.—The secondary eye-vesicle, with lens-fossette.
(After VON KÖLLIKER.)

From a human embryo of four weeks (8 millimetres long). *1*, Mesoderm. *2*, Ectoderm. *3*, Lens-fossette. *4*, Retina. *5*, Pigment layer.

depression. The latter increases, while at the same time its borders approach and finally approximate and coalesce, now forming the sac of the lens. Simultaneously the lateral wall of the primary vesicle yields to this pressure, so that together with the depression it is transformed into a cup-like structure with double walls, through which connective tissue and blood-vessels grow into the cup. Thus the vitreous body, and later

Fig. 3.—Constriction of the lens. (After VAN BAMBEKE.)

Human embryo of four weeks. *1*, Mesoderm. *2*, Pigment layer. *3*, Retinal layer. *4*, Vitreous.

the arteria centralis retinæ, are formed. The cup closes at a later stage of development. At first a defect—the fetal eye-cleft—remains, which, under abnormal conditions, can persist into later life.

For some time the external and internal layers of the eye-cup are separated by a space which, by way of the eye-stalk canal, connects with the third vesicle. Later they are found closely approximated, and conse-

quently the eye-stalk is transformed into a solid mass with the arteria centralis retinæ in its centre. Thus the optic nerve is formed.

Aside from the formation of the vitreous, the surrounding mesoderm contributes, by its development, to the structure of the eye, giving origin to the enveloping membranes, the chorioid and the sclera. It is now necessary to examine more in detail the development of the essential parts of the eye.

The Lens.—The lenticular sac, after separating entirely from the ectoderm, is formed by two or three layers of epithelial cells. Externally it is surrounded by a thin membrane, the capsule, the origin of which is yet doubtful. Soon the anterior and posterior walls show differences in the shape of their cells. The anterior cells gradually transform into other elements, the epithelium lentis, while the posterior ones grow considerably in length. They form a protrusion into the cavity of the sac and change into

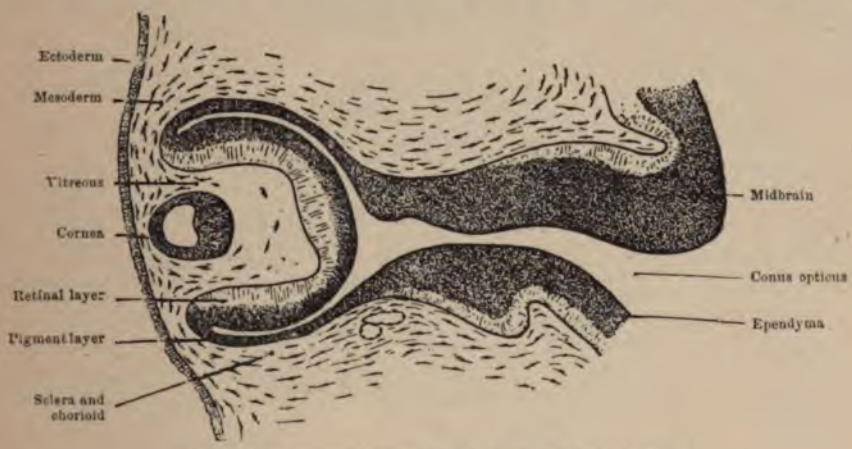


Fig. 4.—The eye-stalk and the eye. (KOLLMANN.)
Human embryo (10.2 millimetres long).

lens-fibres; finally the cavity is obliterated, the fibres reaching the opposite epithelium. The later growth of the lens is obtained by apposition, new fibres being evolved in a formative epithelial zone around the equator of the lens. These new fibres are arranged in concave layers, like the scales of an onion. While soon after birth the metabolism of the lens is only slight, and is achieved by osmosis, active fetal development necessitates a special nutritive apparatus, the tunica vasculosa lentis, around the capsule of the lens. This is a membrane richly provided with blood-vessels. At the time of birth this membrane disappears. Rarely it persists in part, giving rise to the condition known as congenital atresia of the pupil.

The Vitreous Humor.—The formation of the vitreous body begins with the inferior depression of the primary eye-vesicle, commencing at the lens and extending down on the stalk. While in the adult the vitreous does not possess vessels, it is provided with them during fetal life. They arise

from the arteria centralis retinæ, and extend to the posterior surface of the lens, where they spread in the tunica vasculosa and pass over to the anterior surface (Fig. 5). They disappear at birth, only a vestige remaining in the shape of the hyaloid canal.

The account given of the development of the vitreous body is the one which has long been accepted. It is proper to state, however, that Rabl and Tornatola consider the vitreous more essentially ectodermal than mesodermal in nature, and that it is formed from the outer layer of the primitive retina. Lenhossék states that the fibrillary portion of the vitreous is undoubtedly ectodermal in origin, being formed by outgrowths of the cells of the lens, and not from the cells of the inner layer of the retina.

The Secondary Eye-cup and the Eye-membranes.—In mammals the cornea is a product of the mesenchyma. Soon after the separation of the lenticular sac from the ectoderm a thin layer of mesenchyma is found between them, which by migratory cells greatly increases in thickness.

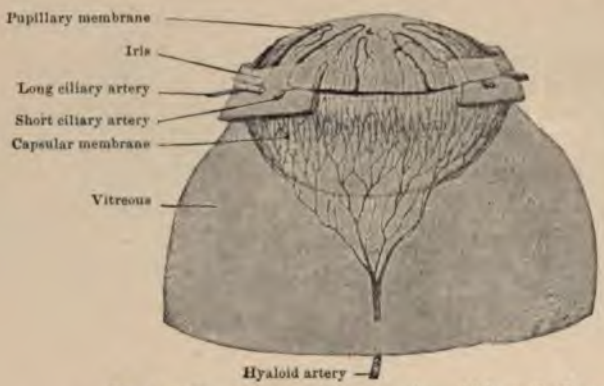


Fig. 5.—Tunica vasculosa lentis. (After O. SCHULTZE.)

The pupillary membrane of a human fetus of eight months; the capsular membrane of a human embryo of six months. A piece of the uvea has been removed.

The inner layer, adjoining the capsule of the lens, represents the pupillary membrane (membrana vasculosa); the external one forms the cornea. These two layers become well defined only when, by a cleft between them, the anterior chamber begins to form. At the same time the external and the internal layers of the eye-cup assume different properties. The external remains thin and is converted into a pigmented lamella by the deposition due to an active proliferation of its cells. A differentiation also is seen between the base of the cup and its rim, the former building up the retina, the latter actively participating in the formation of the ciliary body and the iris. The rim of the cup becomes skin and undergoes a considerable surface growth, squeezing itself in between the cornea and the anterior surface of the lens, where it leaves a small opening—the pupil. Like in the external layer, in this layer also pigment is deposited, which becomes the pigmented layer of the iris. The adjoining layer of the mesenchyma

furnishes the muscular and connective-tissue stroma of the iris. As to the ciliary body, it suffices to say that it takes its origin equally from the thinned marginal zone in conjunction with the attached mesenchymal tissue. In the later stages of development (fourth month) firm connections between this body and the capsule of the lens are produced by connective-tissue proliferation of the former, the zonula of Zinn. The devel-

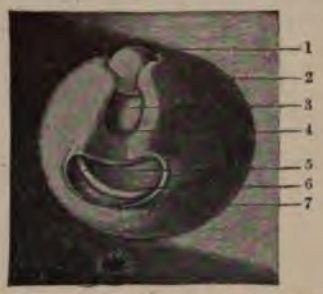


Fig. 6.—Scheme of the secondary eye-vesicle. (MANZ-ZIEGLER.)

1, The lens. *2*, External layer. *3*, Eye-cleft. *4*, Space for the vitreous body. *5*, Primary cavity of the eye-vesicle. *6*, Inner layer of the secondary eye-vesicle. *7*, Wall of the hollow opticus (eye-stalk). The ventral side is turned toward the observer. The stalk of the optic nerve has been cut close to the eye.

opment of the different layers of the retina must be passed by here. Early in ocular development the mesenchymatous layer around the eye-cup differentiates into the two membranes, the chorioid and the sclera.

The Optic Nerve.—Owing to the two depressions formed in the primary eye-cup, the eye-stalk is connected with both layers of the retina. The external layer continues into the pigmented epithelium, the internal

Fig. 7.—Vertical section of the anterior half of an eye from a human embryo of eight or nine weeks. (After VON KÖLLIKER.)

into the retina. The originally hollow eye-stalk gradually becomes a solid mass, the optic nerve, which in its distal end embraces the arteria centralis retinæ. The way in which its primarily spindle-shaped cells transform into the final condition, in which especially the formation of the nerve-fibres is produced, is yet much discussed. Externally the mesenchyma forms two sheaths, one of which is continuous with the pia, while the other merges centrally into the dura and distally into the sclera.

Development of the Retina.—The retina takes its origin from the internal layer of the eye-cup, while the external layer is transformed into the pigment-membrane. Soon after the formation of the secondary eye-cup, the inner layer, by an active proliferation of its cells, begins to thicken, and gives rise to two kinds of cells, the spongioblasts and the neuroblasts.

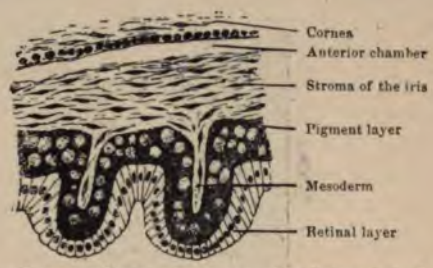


Fig. 8.—Section of two ciliary processes of a human embryo, enlarged 200 diameters. (After KRISCHEWSKI.)

While the former, by peculiar metamorphosis, provide the supporting structures of the retina (Müller's fibres), and also form the two layers called the membrana limitans externa and interna, the latter are the source of the various nerve-cells found in the retina. A small supply of mesoblastic tissue furnishes the necessary amount of vascular structures (arteria centralis retinæ). Of the nerve-cells, those near the pigment-membrane

Fig. 9.—Eye of a human embryo of seven weeks, enlarged 20 diameters. (KOLLMANN.)

1, 2, Limit of the conjunctival sac. *3*, Opening for the ear.

undergo characteristic changes, forming processes which perforate the membrana limitans externa, and later on appear as the layer of rods and cones. While these, therefore, lie externally to the said membrane, the nucleated cell-bodies remain on its inside, and appear as the external nuclear layer. Their axis-cylinder processes take their course toward the centre of the eyeball. The remaining part of the retinal nerve-cells gives

rise to the other layers, some becoming the bipolar cells of the inner nuclear layer and others forming the large ganglion-cells of the ganglion-cell layer. All of them converge toward the papilla, where they pass through the retina and the two connective-tissue sheaths of the eye-cup to appear as optic-nerve fibres. The sensory epithelium is formed latest in the course of development. In some mammals (cats and dogs) it appears only after birth.

The part of the retinal layer from the ora serrata to the pupillary opening is not differentiated like the retina in the fundus of the eye. Its two divisions, the lenticular and the marginal zone, enter into the formation of the ciliary body and iris.

The manner of formation of the optic-nerve fibres is still in dispute. Some observers claim that they arise from the ganglion-cells of the thalami optici and the anterior corpora quadrigemina, while others maintain that they are nothing but the prolonged axis-cylinders of the retinal ganglion-cells.

A well-developed area centralis is seen in the fifth or sixth month, while the formation of the fovea does not occur until the eighth month. The distribution of the capillary system from the arteria centralis is complete by the sixth month.

Accessory Organs of the Eye.—The upper and lower eyelids are formed by two folds of the external skin, which grow from above and below over the cornea until they meet. In many mammals, and so, too, in man, they coalesce with their epithelial margins (third month). Shortly before birth they again separate. By these two folds the conjunctival sac is formed. At the time of coalescence, by proliferation of the rete Malpighii, the glands of Meibomius are seen to grow into the lids as solid, finger-like projections, on which small lateral buds develop later. The solid glands acquire lumina by the central cells undergoing fatty degeneration and solution. Simultaneously cilia arise, the development of which does not differ from that of other hair-follicles. The lacrimal gland appears during the third month. It is formed by epithelial proliferation of the conjunctival sac at the outer part of the eye, where the upper lid merges into the eyeball. As to the lacrimal canal, it must suffice to say that it originates very early from a solid strand of cells in which a lumen forms. The same is true of the canaliculi.

CHAPTER II.

ANATOMY OF THE EYE.

THE ORBIT.

THE eye is placed in the bony cavity known as the orbit. (The orbits are two in number and are situated at the upper and anterior part of the face.) Each orbit is pyramidal in shape, and presents a base, an apex, four sides, and four angles, and is composed of seven bones, viz.: frontal,

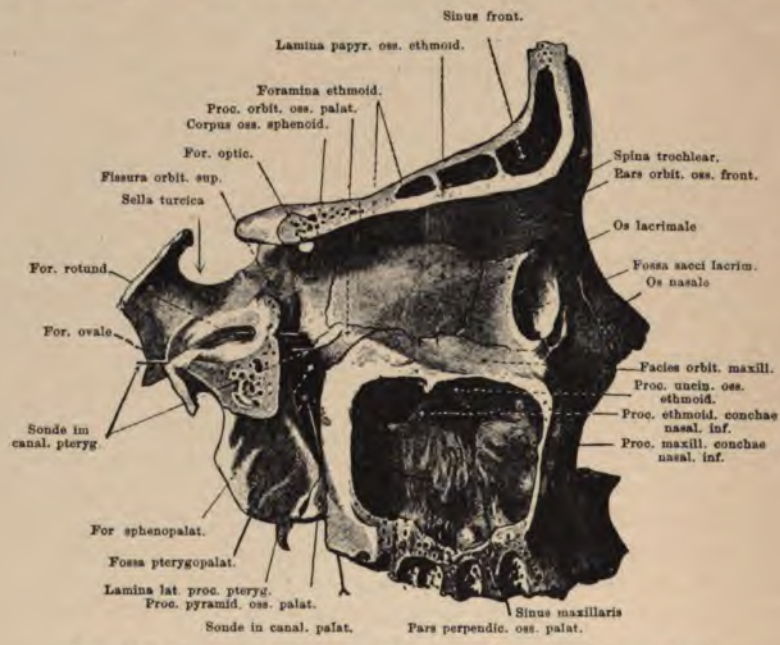


Fig. 10.—Inner wall of the orbit and adjacent parts. (His.)

sphenoid, ethmoid, superior maxillary, malar, palate, and lacrimal. Three of these—the frontal, sphenoid, and ethmoid—enter into the formation of both orbits. Hence, while each orbit is composed of seven bones, the two are made up of eleven bones only. The apex is directed upward and inward, the base downward and forward. If continued, the axes of the orbits would cross in the region of the sella Turcica of the sphenoid. The roof of the orbit is formed by the orbital plate of the frontal bone in front, and the lesser wing of the sphenoid behind. It is concave, and at its external and anterior part presents a depression, the lacrimal fossa, which marks the site of the lacrimal gland. The roof is always thin and

sometimes is wanting in part. In such instances the dura mater and periorbita are in contact. Internally and anteriorly the roof presents a depression, the trochlear fossa, for the pulley of the superior oblique muscle. The floor slopes downward, forward, and outward, and is made up of the orbital plate of the superior maxilla, the orbital process of the malar, and the orbital plate of the palate bone. The inner wall is formed from the nasal process of the superior maxilla, the lacrimal, ethmoid, and sphenoid bones. The outer wall is formed from the orbital process of the malar and the great wing of the sphenoid. Each orbit is in relation with the cranial cavity above, the antrum of Highmore below, the temporal fossa externally, and the nasal cavity, frontal, ethmoidal, and sphenoidal sinuses internally. The depth of the orbit is one and three-fourth inches and is much increased by the addition of the soft parts. Foreign bodies of large size have lodged in the orbit, where their presence was unsuspected. The orbit is lined by periosteum derived from the dura mater. This is attached closely to the optic foramen and sphenoidal fissure behind, and to the margin of the orbit in front, where it is continuous with the periosteum covering the bones of the face. The walls of the orbit in front form a strong bony ring, the orbital margin. This protects the eye against external force, particularly above and below, where the margin projects farthest. Internally there is no well-defined orbital margin, but here the nose protects the eye. Externally the margin recedes, and here the eye is most exposed to injury.

Foramina.—Nine foramina communicate with each orbit. They are the optic foramen, sphenoidal fissure, malar canals, anterior and posterior ethmoidal, supra- and infra-orbital foramina, spheno-maxillary fissure, and the canal for the nasal duct. The *optic foramen*, placed above the level of the middle of the eye, is a round opening at the apex of the orbit, situated between the two roots of the lesser wing of the sphenoid bone. It transmits the optic nerve and the ophthalmic artery. The *sphenoidal fissure*, or foramen lacerum anterius, also situated at the apex, is a slit-like opening between the greater and lesser wings of the sphenoid. It transmits the third, fourth, and sixth nerves; the frontal, nasal, and lacrimal branches of the ophthalmic, or first division of the fifth, nerve; filaments from the cavernous plexus of the sympathetic nerve, the orbital branch of the middle meningeal artery, the recurrent lacrimal artery, and the ophthalmic vein.

The *malar*, or zygomatico-temporal *canals*, are small openings which run from the orbital plate of the malar bone to the external surface of the same, and serve for the passage of nerves and vessels. The *ethmoidal foramina* are two grooves in the upper surface of the os planum. They are converted into foramina by the articulation of the frontal bone with the ethmoid. The anterior ethmoidal foramen transmits the nasal nerve and anterior ethmoidal artery; the posterior ethmoidal foramen gives passage to the posterior ethmoidal artery. The *supra-orbital foramen*, often only

a notch, is situated in the supra-orbital margin of the frontal bone at the junction of the inner with the middle third. It can be felt in the living subject, and transmits the supra-orbital vessels and nerve. The *infra-orbital foramen,* which opens upon the facial surface of the superior maxilla, begins as a groove in the orbital surface of the superior maxillary bone. It passes forward and terminates in a canal which has two branches. One of these ends in the infra-orbital foramen; the other runs in the anterior wall of the antrum and is called the anterior dental canal. It transmits the anterior dental vessels and nerve to the front teeth of the upper jaw. The infra-orbital foramen is for the passage of the infra-orbital vessels and nerve. The *spheno-maxillary fissure* forms the external boundary of

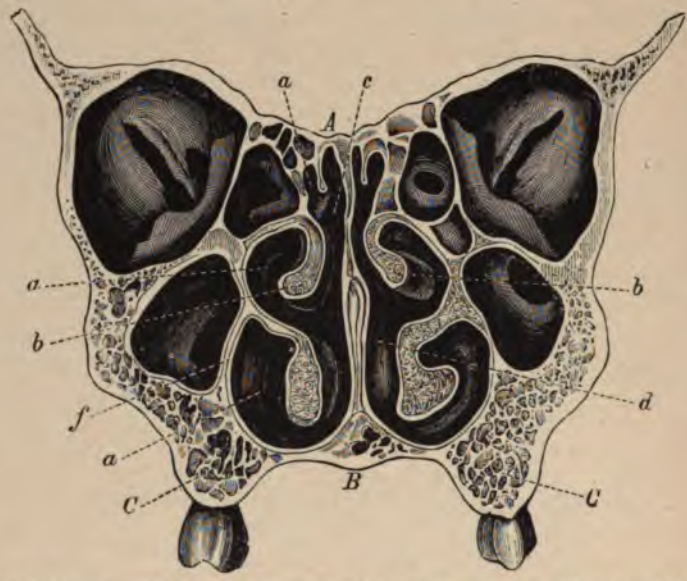


Fig. 11.—Frontal section showing the relation of the orbits to the other cavities of the skull. (After ZUCKERKANDL.)

A, Roof of interorbital space. *a*, *a*, *a*, The three nasal meati. *b*, Middle turbinated bone. *c*, Olfactory fissure. *d*, Respiratory region. *B*, Floor of the nasal chambers.

the floor of the orbit. It is formed chiefly by the orbital plate of the superior maxillary bone, with a small part of the malar in front and the orbital plate of the palate bone behind. It transmits the superior maxillary nerve and its orbital branch, the infra-orbital vessels, and ascending branches from Meckel's ganglion. By means of the spheno-maxillary fissure the orbit communicates with three fossæ: the temporal, zygomatic, and spheno-maxillary. The *canal for the nasal duct* is at the inner and anterior part of the orbital floor. It begins with the lacrimal groove, a depression formed by the lacrimal bone and the nasal process of the superior maxilla. The canal runs downward, outward, and backward, and opens into the inferior meatus of the nose, beneath the anterior part of the inferior turbinated bone.

Interorbital Space.—This includes the nasal and lacrimal bones, the ascending processes of the superior maxillary bones, the frontal below the level of the supra-orbital foramina, and the lateral masses and perpendicular plate of the ethmoid bone.

Orbital Contents.—The orbit contains periosteum, adipose and connective tissue, the eyeball, muscles, vessels, nerves, glands, and ganglia. These form a soft cone, which fits accurately into the orbit.

THE PERIOSTEUM is derived from the dura mater. The dura is closely attached to the sphenoidal fissure and optic foramen, in front of which it divides into two layers. The outer one forms the periorbita and the inner divides the orbital contents into different compartments. This layer will be described under the name of the capsule of Tenon. The periorbita runs forward to the base of the orbit. It is closely attached to the orbital margin, and disappears in the periosteum covering the bones of the face. It is loosely connected with the walls of the orbit. Hence, in

Fig. 12.—Capsule of Tenon. (MERKEL.)

fracture with effusion of blood or in cases of purulent collection, this membrane may be extensively removed from the bone.

THE CAPSULE OF TENON, or oculo-orbital fascia, insheathes all the organs which pass through it, forms an acetabulum in which the eyeball rests, is continuous with the sheath of the optic nerve, forms a secondary attachment for the ocular muscles, and prevents morbid collections in the orbit from reaching the surface. It covers the posterior four-fifths of the eyeball (except a space one centimetre in diameter in the middle of which the optic nerve passes) and here consists of two layers, between which is *Tenon's space.* This is continuous with the subdural and subarachnoidal spaces of the optic nerve. The capsule of Tenon divides and subdivides, forming a covering for every tissue in the orbit. Some anatomists limit Tenon's capsule to that part of the fibrous envelope of the eyeball in front of the point where it is pierced by the ocular muscles, and they give the name of Bonnet's capsule to the part behind. Tenon's space, according to Schwalbe, is a lymph-space; but this is denied by Langer. That part of the dura which surrounds the optic nerve from its entrance through

the optic foramen to the posterior surface of the eyeball is known as *Bonnet's sheath,* or the dural covering of the nerve.

The Orbital Muscles are seven: four recti, two obliqui, and the levator of the upper lid. All of them, except the levator, are attached to the eyeball. The levator is attached to the tarsal plate of the upper eyelid. All except two of the muscles are supplied by the motor oculi nerve. The exceptions are the superior oblique and the external rectus. The former is controlled by the patheticus, the latter by the abducens nerve. All of the orbital muscles except the inferior oblique arise from the bone around the optic foramen and run forward to be attached to the eyeball at different distances from the cornea. The muscles pierce the capsule of Tenon in front of the equator, and they receive sheaths from the capsule. The sheath becomes inseparable from the tendon on the ocular surface or side of

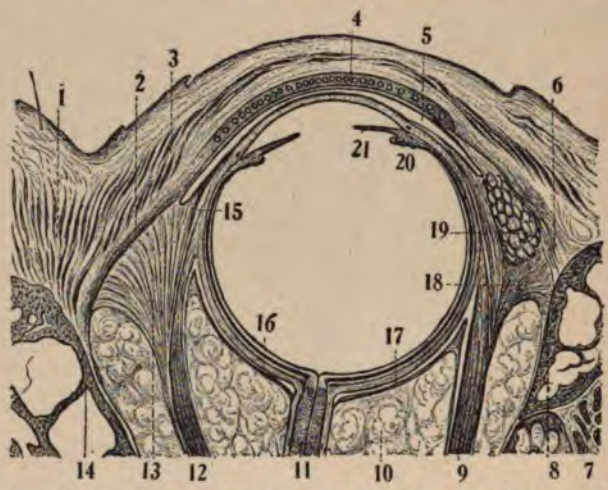


Fig. 13.—Horizontal section of the right orbit, viewed from above. (After Gerlach.)

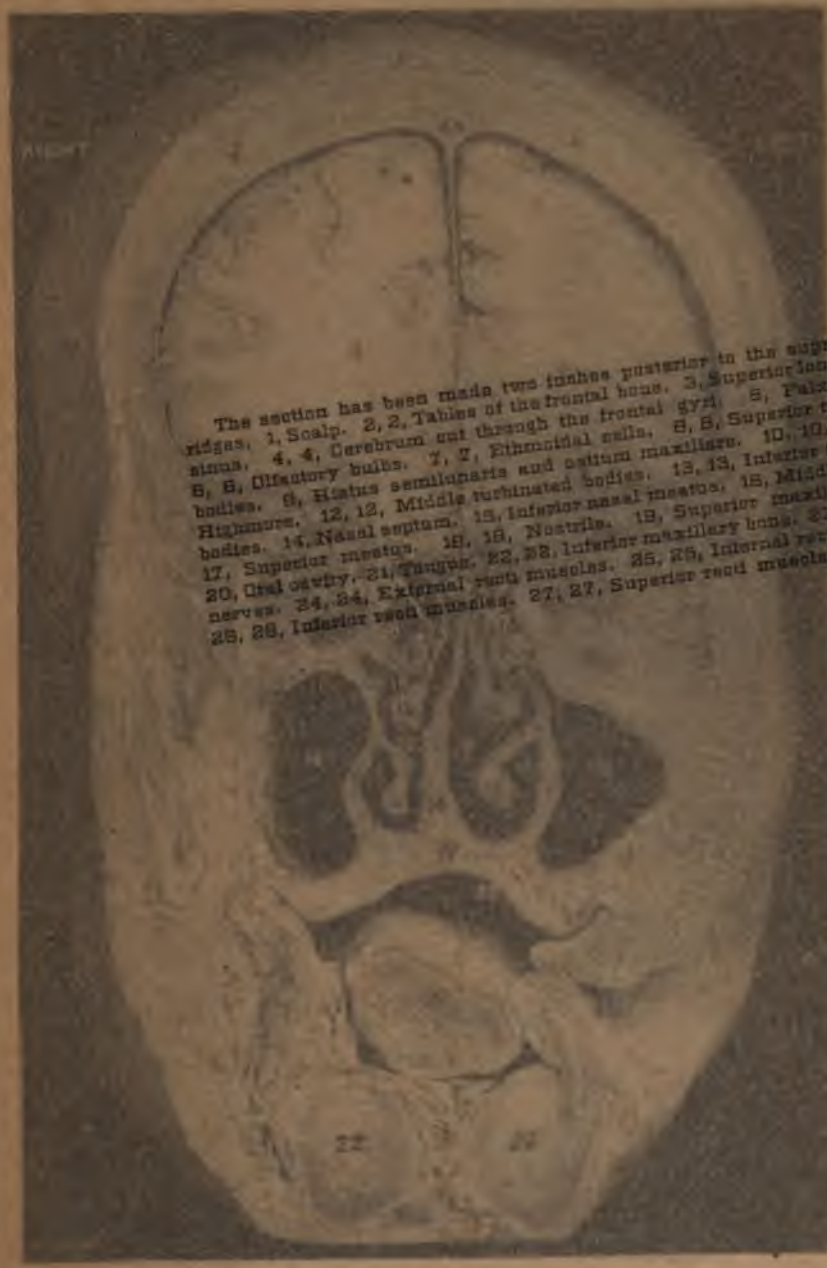
1, Horner's muscle. *2*, Septum orbitale. *3*, Fibres of the orbicularis palpebrarum muscle. *4*, Tarsal plate. *5*, Conjunctival sac. *6*, Outer palpebral ligament. *7*, Temporal muscle. *8*, Wall of the orbit. *9*, External rectus muscle. *10*, Orbital fat. *11*, Optic nerve. *12*, Internal rectus muscle. *13*, Inner check ligament. *14*, Inner wall of the orbit. *15*, Attachment of the capsule of Tenon to the conjunctiva. *16*, Capsule of Tenon. *17*, Tenon's space. *18*, Outer check ligament. *19*, Lacrimal gland. *20*, Ciliary process. *21*, Iris.

pressure, but on the orbital surface it is commonly separable. According to numerous measurements made by Fuchs, the insertion of the internal rectus is 5.5 millimetres from the cornea; of the external rectus, 6.9 millimetres; of the superior rectus, 7.7 millimetres; of the inferior rectus, 6.5 millimetres; of the superior oblique, 16 millimetres; and of the inferior oblique, 17.3 millimetres. Unstriped muscular fibres radiating from the deep margin of the tarsus to the orbit have been named *Müller's muscle.* They are more numerous in the upper than in the lower eyelid. Fibres of the capsule of Tenon pass from the anterior extremity of each muscle and find attachment in the sclera. It is for this reason that after the

PLATE I.

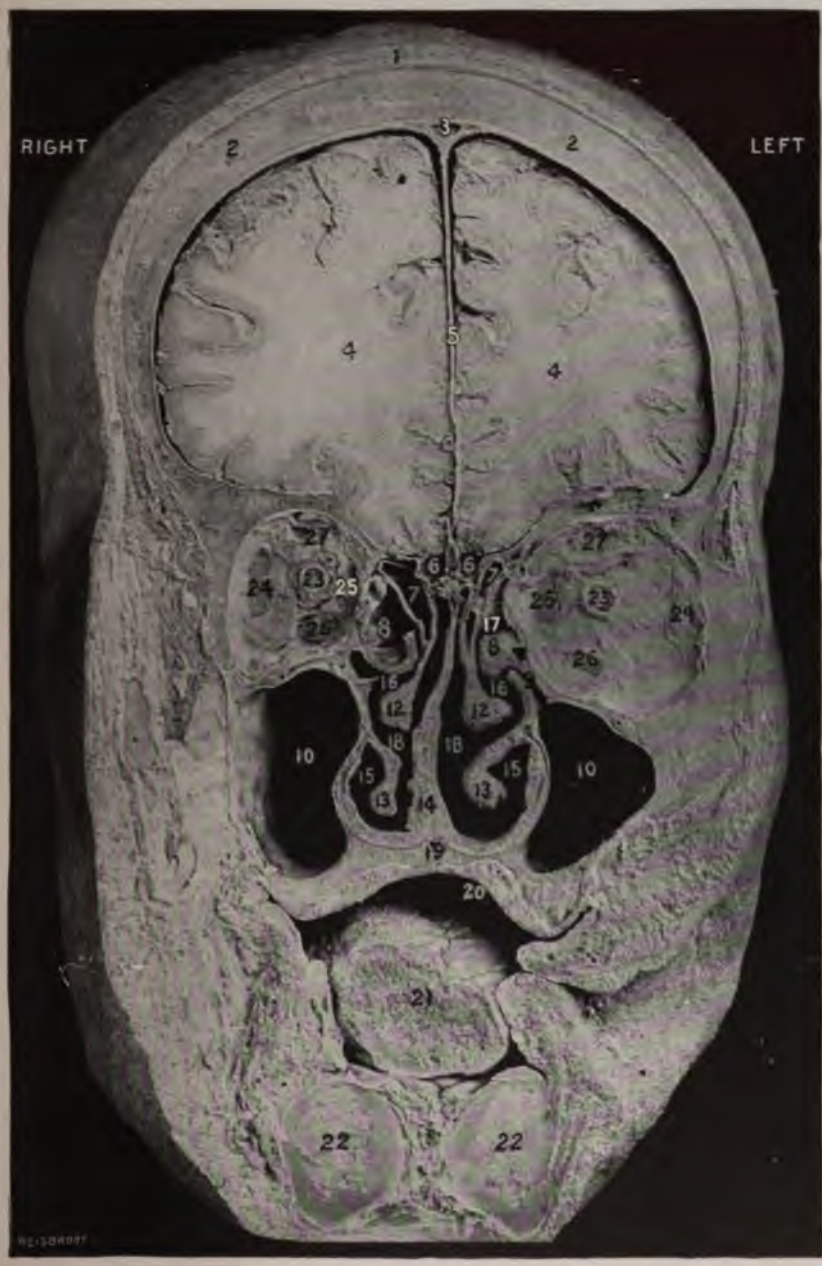
Coronal Section Showing the Orbits and Adjacent Parts.

(Courtesy of Dr. John W. Murray, of Cincinnati.)



The section has been made two inches posterior to the supra-orbital ridges. 1, Scalp. 2, 2, Tables of the frontal bone. 3, Superior longitudinal sinus. 4, 4, Cerebrum cut through the frontal gyri. 5, Falx cerebri. 6, 6, Olfactory bulbs. 7, 7, Ethmoidal cells. 8, 8, Superior turbinated bodies. 9, Hiatus semilunaris and ostium maxillare. 10, 10, Antra of Highmore. 12, 12, Middle turbinated bodies. 13, 13, Inferior turbinated bodies. 14, Nasal septum. 15, Inferior nasal meatus. 16, 16, Inferior turbinated 17, Superior meatus. 18, 18, Nostrils. 19, Superior maxillary bone. 20, Oral cavity. 21, Tongue. 22, 22, Inferior maxillary bone. 24, 24, External optic nerves. 25, 25, Internal recti muscles. 27, 27, Superior recti muscles. 28, 28, Inferior recti muscles.

Fig. 18.—Horizontal section of the right orbit, viewed from above.


RIGHT
LEFT
1
2
2
3
4
4
5
6
6
7
7
8
8
10
10
12
12
13
13
14
15
15
16
16
17
18
18
19
20
21
22
22
23
23
24
24
25
25
26
26
27
27

operation of tenotomy the affected muscle is able to exert an active, although reduced, influence. After tenotomy the reattachment of the muscle is more by the capsule of Tenon and the conjunctiva than by the tendon. The *levator palpebræ superioris* arises from the common origin above the rectus superior, passes forward, becomes fleshy, widens out to form an elongated triangle, and is inserted on the anterior surface of the upper tarsal plate. A few fibres are inserted into the sheath of the palpebral muscle and skin of the upper lid. The levator is in relation above with the frontal nerve, supra-orbital artery, and periorbita; below with the rectus superior and upper lid. It is sometimes absent, producing congenital ptosis. The

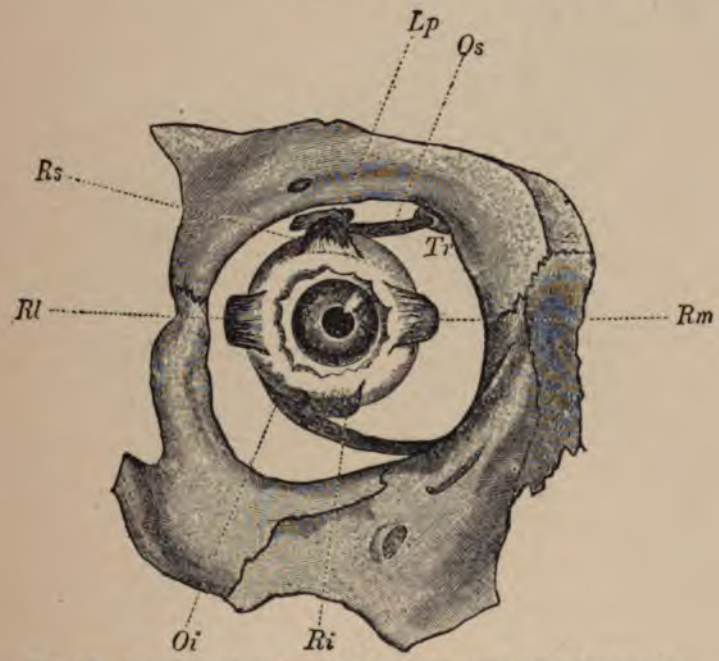


Fig. 14.—Position of eyeball in the right orbit and the location of the orbital muscles. (MERKEL.)

Lp, Levator palpebræ superioris. *Os*, Superior oblique. *Rs*, Superior rectus. *Rl*, External rectus. *Oi*, Inferior oblique. *Ri*, Inferior rectus. *Rm*, Internal rectus. *Tr*, Trochlea.

rectus superior, the weakest of the recti muscles, arises from the upper margin of the optic foramen, and passes forward beneath the levator. It is in relation with the levator above, and sends a small muscular bundle to it. Below, it lies upon the orbital fat, which surrounds the optic nerve, and is also in relation with the nasal nerve, ophthalmic artery and vein, the capsule of Tenon, and the globe of the eye. The *superior oblique* extends from the common origin to the internal angular process, where it becomes tendinous, passes through a pulley, and bends suddenly backward, outward, and downward at an angle of 30 degrees. The tendon passes beneath the superior rectus muscle, and is inserted into the posterior part of the sclera,

on a level with the equator of the eye, and midway between the superior and external recti muscles. The superior oblique is in relation above with the periorbita and fourth nerve; the tendon is in relation with the superior rectus. Below, the muscle is in relation with the nasal nerve and upper border of the internal rectus. The *trochlea* is a curved tubular pulley of hyalin cartilage fastened by fibrous tissue to the trochlear fossa of the frontal bone. A true synovial membrane is rarely found lining the trochlea. The reflected tendon, nearly twenty twenty-fifths of an inch long, is covered with a loose sheath derived from Tenon's capsule. The *external rectus* arises by two heads, between which pass the motor oculi nerve, the nasal

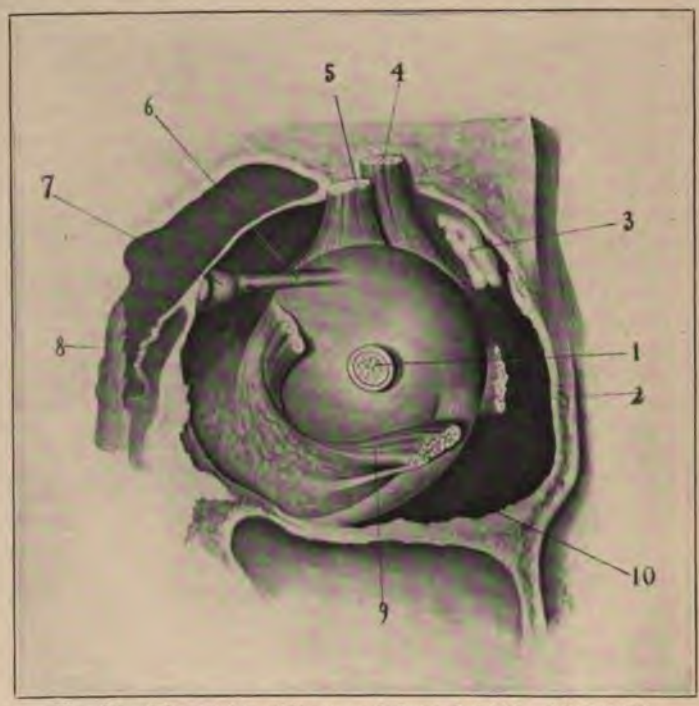


Fig. 15.—The right orbit opened from behind. (AUTHOR.)

(Original drawing by DR. R. W. MILLS.)

1, Optic nerve. *2*, External rectus. *3*, Lacrimal gland. *4*, Levator palpebræ superioris. *5*, Superior rectus. *6*, Superior oblique. *7*, Trochlea. *8*, Internal rectus. *9*, Inferior oblique muscle.

branch of the ophthalmic division of the fifth nerve, the sixth nerve, and the ophthalmic vein. The muscle passes forward, diverging at an angle of 60 degrees from its fellow in the opposite orbit, and is inserted into the sclera. It is in relation externally with the periorbita; internally, with the fatty cone, capsule of Tenon, ciliary ganglion, ophthalmic artery, and nasal nerve. The *rectus internus*, the strongest of the series, passes from the optic foramen forward, being nearly parallel with its fellow, and is inserted into the sclera by a tendon which is concave forward. Its upper edge is in relation with the nasal nerve and ethmoidal vessels. The *rectus*

inferior, the shortest of the series, passes from the margin of the optic foramen to the sclera, and is attached at a point midway between the internal and external recti. The capsule of Tenon covering its tendon gives off slips to the lower lid in front of the tarsal plate and to the lower fornix. This muscle is in relation with the optic nerve, fatty cone, ciliary ganglion, and ciliary nerves above, while below adipose tissue separates it from the floor of the orbit. The *inferior oblique,* the only orbital muscle which does not arise from around the optic foramen, takes its origin from the orbital

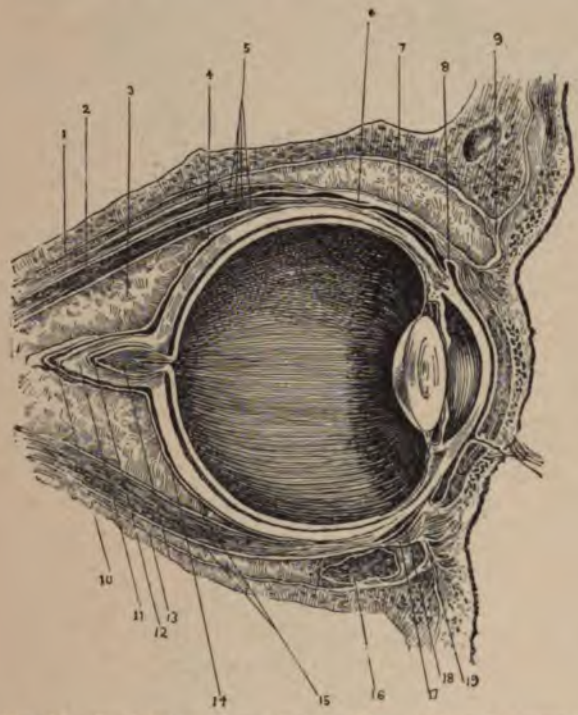


Fig. 16.—Sagittal section of the orbit. (After DEAVER.)

1, Levator palpebræ superioris. *2,* Superior rectus. *3,* Orbital fat. *4,* Capsule of Tenon. *5,* Orbital fascia. *6,* Connection between the superior rectus and levator palpebræ superioris. *7,* Capsule of Tenon. *8,* Fornix of the conjunctiva. *9,* Septum orbitale, or orbito-tarsal ligament. *10,* Supravaginal lymph-space. *11,* Dural sheath of optic nerve. *12,* Intervaginal lymph-space. *13,* Periosteum of orbit. *14,* Capsule of Tenon. *15,* Orbital fascia. *16,* Inferior oblique muscle. *17,* Check ligament of inferior rectus muscle. *18,* Capsule of Tenon. *19,* Orbito-tarsal ligament.

plate of the superior maxillary bone at a point just external to the lacrimal groove. Its small, fleshy belly passes backward, outward, and a little upward, to be inserted into the sclera between the external and superior recti, and slightly nearer the optic nerve than the insertion of the superior oblique. As it lies in the orbit it is a flat band whose surfaces look upward and downward, respectively.

CHECK LIGAMENTS.—Near the insertions of the recti muscles the anterior layer of the orbital fascia forms strong, band-like processes called

check ligaments. They extend laterally from the external rectus and internal rectus to the malar and lacrimal bones, respectively (*18*, *13*, in Fig. 13). Superiorly a similar band connects the superior rectus muscle

Fig. 17.—Dissection of base of left orbit. (AUTHOR.)

(Original drawing by DR. R. W. MILLS.)

This shows the insertion of the levator palpebræ superioris, the lacrimal gland, pulley of the superior oblique muscle, and orbital fat.

with the levator palpebræ (*6*, in Fig. 16). Inferiorly a fibrous band passes from the rectus inferior to a process from the obliquus inferior muscle, the conjoined band being attached to the floor of the orbit. The check

Fig. 18.—Distribution of the third and sixth nerves in the orbit. (LÉVEILLÉ.)

1, The third nerve. *2*, Its superior division. *3*, Its inferior division. *4*, Branch to the inferior oblique muscle. *5*, The sixth nerve, distributed to the external rectus muscle.

ligaments prevent extreme muscular action, and after tenotomy prevent deep retraction of the muscles. By acting on the posterior hemisphere of Tenon's capsule they oppose excessive backward traction on the part

of the recti muscles. In addition to the check ligaments, other connective-tissue fibres pass in an irregular manner from one muscle to another (Howe).

NERVES OF THE ORBIT.—The nerves contained in the orbit are the optic, motor oculi, patheticus, three branches of the ophthalmic division of the fifth, the abducens, and filaments from the cavernous plexus of the sympathetic. The optic nerve will be described later.

The *Motor Oculi*, or third nerve, enters the orbit via the sphenoidal fissure, where it has already divided into two branches: a superior and an inferior. The former supplies the levator palpebræ and rectus superior, while the latter is distributed to the internal and inferior recti muscles and to the inferior oblique. The inferior branch also is distributed to the ciliary muscle and iris through the ciliary ganglion. The motor oculi

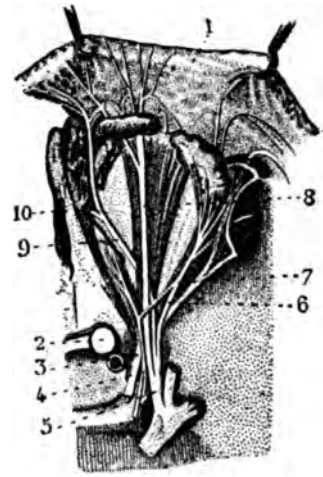


Fig. 19.—Ophthalmic division of the fifth nerve. (LÉVEILLÉ.)

1, Skin of the forehead, turned down. *2*, Optic nerve. *3*, Third nerve. *4*, Fourth nerve. *5*, Ophthalmic division of the fifth nerve. *6*, Lacrimal branch. *7*, Union of the fourth nerve with the lacrimal branch of the fifth. *8*, Frontal. *9*, Nasal. *10*, Internal branch of nasal.

supplies all the muscles of the orbit except two: the superior oblique and the external rectus. The motor oculi contains about 15,000 fibres, which are distributed to five muscles containing about 40,000 muscular fibres.

The *Ciliary, or Lenticular, Ganglion*, sometimes called the ophthalmic ganglion, is a small quadrate body about the size of a pin's head. It is placed at the back part of the orbit internal to the external rectus muscle. It can be found by tracing the branch of the third nerve to the inferior oblique backward, when the ganglion will be seen. The ganglion receives the long root of the nasal nerve at its upper posterior angle; into its lower posterior angle the short, thick root passes from the nerve to the inferior oblique; and several fine filaments from the cavernous plexus of the sympathetic enter its posterior border. The branches from the ciliary ganglion are given off anteriorly, and are about six in number, called the

short ciliary nerves. These pass forward in the perineural space, accompanying the short ciliary arteries, and each branch divides into several, so that there are about twenty nerves. They pierce the sclera, pass forward in delicate grooves on its inner surface, and are distributed to the ciliary muscle, iris, and cornea. Before reaching the eyeball they are joined by filaments from the nasal nerve. The branches of communication of the ciliary ganglion are: the motor branch from the motor oculi; the sensory, from the nasal nerve; and the sympathetic, from the cavernous plexus.

The *Fourth, or Patheticus, Nerve,* the smallest of the cranial nerves, enters the orbit via the sphenoidal fissure, and is distributed to the superior oblique muscle on its upper surface. It consists of about 2000 coarse fibres, which innervate a muscle having about the same number of fibres.

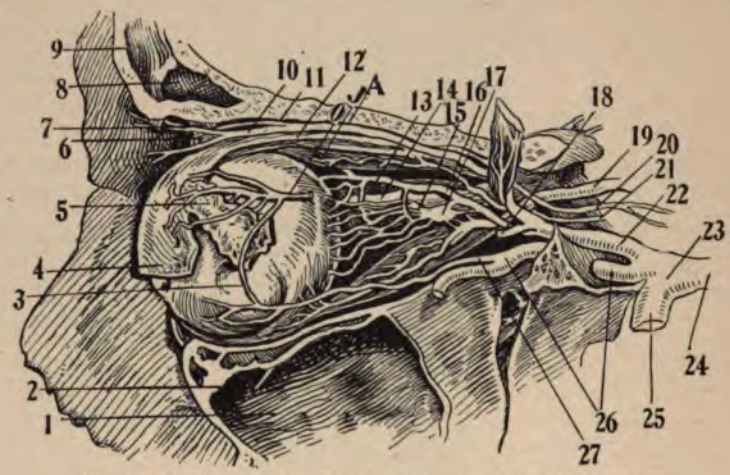


Fig. 20.—Lateral view of the orbit, showing the nerves.
(After DEAVER.)

1, Antrum. *2*, Bristle in the antrum. *3*, Loop between orbital and lacrimal nerves. *4*, Tarsal plate. *5*, Lacrimal gland. *6*, Tendon of superior oblique. *7*, Pulley of the same. *8*, Infundibulum. *9*, Frontal sinus. *10*, Supra-orbital nerve. *11*, Supratrochlear nerve. *12*, Levator palpebræ muscle. *O*, Lacrimal nerve. *J*, Superior rectus muscle *A*, Frontal nerve. *13*, Internal rectus muscle. *14*, Optic nerve. *15*, Short ciliary nerve. *16*, Nasal nerve. *17*, Ciliary ganglion. *18*, Lacrimal nerve. *19*, Motor oculi nerve. *20*, Patheticus nerve. *21*, Abducens nerve. *22*, Ophthalmic division of fifth nerve. *23*, Gasserian ganglion. *24*, Fifth nerve. *25*, Inferior maxillary nerve. *26*, Superior maxillary nerve. *27*, Orbital nerve.

The *Ophthalmic Nerve,* one of the three primary branches of the great fifth nerve, enters the orbit via the sphenoidal fissure, after dividing into the frontal, lacrimal, and nasal nerves. The *frontal nerve* passes between the periorbita and the levator muscle. Immediately behind the margin of the orbit it divides into supratrochlear and supra-orbital branches. The former escapes from the orbit internal to the trochlea and supplies the periosteum, the skin at the root of the nose, and inner part of the upper eyelid. The latter emerges via the supra-orbital foramen and supplies the upper eyelid, periosteum of the forehead, and scalp. The *lacrimal nerve* sends branches to the lacrimal gland, conjunctiva of the external canthus,

and upper eyelid, and gives off an inferior branch, which joins branches of the superior maxillary nerve. Branches from the resulting arc supply the lacrimal gland. Stimulation of either stem of this loop causes lacrimation. Division results in the pouring out of a paralytic secretion. The *nasal nerve* enters the orbit between the heads of the external rectus, passes obliquely across the orbit, enters the anterior ethmoidal foramen, passes between two fronto-ethmoidal cells, enters the cranial cavity, crosses the ethmoidal plate, enters a slit by the side of the crista galli, grooves the inner surface of the nasal bone, and divides into three branches. These are distributed to the nasal mucous lining and the skin as far as the tip of the nose. Before entering the ethmoidal foramen the nerve gives off branches to the ciliary ganglion, and the long ciliary nerves which pierce the sclera and end in the eyeball. These will be described with the eye.

The *Sixth, or Abducens, Nerve* enters the orbit via the sphenoidal fissure between the heads of the external rectus, and supplies that muscle. It neither gives off nor receives branches in the orbit.

Meckel's Ganglion, or the spheno-palatine ganglion, deeply placed in the pterygo-maxillary fossa, gives off orbital branches. These reach the orbit via the spheno-maxillary fissure, and are distributed to the periorbita. It is by means of these that a communication exists between the spheno-palatine and the ciliary ganglia.

The Cervical Sympathetic.—This part of the nervous system is of importance to the ophthalmologist, since all sympathetic fibres passing to the orbit traverse the superior cervical ganglion, except possibly those which follow the course of the fifth nerve. The cervical portion of the nerve consists of three ganglia and the intervening strands.

The *superior cervical ganglion,* the largest of the three, is a fusiform body about an inch in length, and is placed in front of, and internal to, the transverse processes of the second and third vertebræ. It gives branches to the anterior divisions of the first four cervical nerves, to the hypoglossal, one to each of the ganglia of the vagus, one to the petrous ganglion of the glosso-pharyngeal, several to the carotid arteries, the pharynx, several cardiac branches, and branches to the small deep petrosal nerve, the cavernous plexus, the third and fourth nerves, and the ophthalmic division of the fifth. The sympathetic root of the lenticular ganglion passes into the orbit either separately or in conjunction with the nasal branch of the fifth nerve.

The *middle sympathetic ganglion,* placed opposite the sixth cervical vertebra, is smaller than the superior, and is sometimes absent. It gives communicating branches to the fifth and sixth cervical nerves, vascular branches to the inferior thyroid artery and thyroid gland, and a middle cardiac branch.

The *inferior cervical ganglion* lies on the neck of the first rib. It gives branches to the seventh and eighth cervical nerves, to the vertebral artery, and to the deep cardiac plexus.

Blood-vessels of the Orbit.—The *Arteries* of the orbit are derived from the internal carotid by way of the ophthalmic. The *ophthalmic artery* is given off from the cavernous portion of the carotid almost at a right angle: a fact which explains the infrequency of embolism of the ophthalmic or of its branches as compared with the same process in the vessels of the brain. It enters the orbit through the optic foramen, lying below the optic nerve, and gives off ten branches, as follows: (1) lacrimal, (2) posterior ciliary, (3) supra-orbital, (4) central retinal, (5) posterior ethmoidal, (6) anterior ethmoidal, (7) palpebral, (8) muscular, (9) frontal, and (10) nasal. The *lacrimal artery* is the largest branch. It passes above the rectus externus muscle to the lacrimal gland, to which it gives branches. Then it leaves the orbit at the superior external angle and terminates in the external palpebral. The lacrimal sends a recurrent branch through

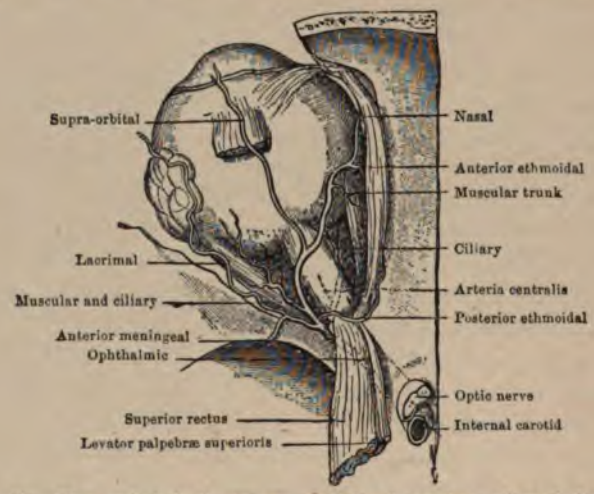


Fig. 21.—The ophthalmic artery and its branches, viewed from above. (After Merkel and Kallius.)

the sphenoidal fissure to anastomose with the middle meningeal artery. Other branches pass to the malar foramina, the periorbita, the external and superior recti, and levator palpebræ muscles. The *posterior ciliary arteries* are of two sets, the short and the long. The *short ciliary arteries*, from four to ten in number, arise from the lacrimal as it crosses the optic nerve. They run forward in a tortuous course surrounding the nerve, and pierce the sclerotic perpendicularly near the optic nerve. Before reaching the sclera each artery divides, so that the short ciliary arteries are from fifteen to twenty in number. The further course of these vessels will be traced in connection with the anatomy of the eyeball. The *long ciliary arteries* are two in number; they also run forward and pierce the sclera. The *supra-orbital artery*, usually a small branch, arises on the inner side of the superior rectus muscle over the optic nerve, passes out of the orbit via the supra-orbital notch or foramen, and ends on the forehead, where

it supplies the occipito-frontalis muscle and the periosteum. In its course it gives off diploïc branches to the frontal sinus and diploë. The *central retinal artery* is a minute vessel which enters the optic nerve from beneath, passes forward in the substance of the nerve, and enters the eyeball, where it supplies the retina. The comes nervi ischiadici and the central retinal are the only arteries in the human subject which traverse the substance of nerves for any considerable distance. The *posterior and anterior ethmoidal* are small vessels which pass to the ethmoidal cells and dura mater. The *muscular branches* are twigs which supply the ocular muscles. The

Fig. 22.—Distribution of the ophthalmic vein. (GURWITSCH.)

frontal artery escapes at the upper internal angle of the orbit and supplies the inner part of the upper lid and the brow. The *nasal artery* pierces the orbicularis palpebrarum muscle over the internal palpebral ligament, passes along the root of the nose to anastomose with terminal branches of the facial artery, and sends branches to the lacrimal sac, canal, and caruncle, and to the vessel of the opposite side.

The Veins of the Orbit are commonly described as two in number: the superior ophthalmic and the inferior orbital veins. The *superior ophthalmic vein,* which forms the largest channel by which blood from the face and head can enter the cavernous sinus, starts from the inner and

upper part of the orbit, where it communicates with the frontal and facial veins, and passes backward to the sphenoidal fissure. On the way it receives branches from the eyeball, the ocular muscles, the ethmoidal cells, and from the inferior orbital vein. This vein is an important factor in many ocular diseases. One of the principal causes of thrombosis of the cavernous sinus is infective inflammation within the orbit, passing backward through the veins. The *inferior ophthalmic vein* lies at the floor of the orbit between the inferior and external recti muscles, and begins in venules derived from the facial and malar veins. Through the spheno-maxillary fissure it communicates with the pterygoid plexus, into which it often empties, although it may end in the superior ophthalmic vein, with which it always anastomoses.

THE INTERMUSCULAR CONE OF FAT.—The interstices between the orbital contents are filled with adipose tissue, and the whole is bound together by fasciæ. Adipose tissue is placed within the muscular cone. Its anterior surface is concave to fit the eyeball and is lined with Tenon's capsule. Adipose tissue is also present outside of the cone, between the muscles and the periorbita.

LYMPH-VESSELS AND LYMPHATICS.—These are wanting in the orbit.

Position of the Eyeball.—The globe is placed not in the axis of the orbit, but below and external to it. The prominence of the eyeball is largely dependent upon the amount of adipose tissue in the orbit; the greater the amount of adipose, the greater is the prominence. In emaciated subjects the eyeballs are sunken from diminution of the fat of the orbit.

The Lacrimal Apparatus.—This consists of the lacrimal gland of the orbit, the accessory lacrimal glands found in the lids, the puncta, canaliculi, sac, and the nasal duct. The *lacrimal gland*, a compound racemose gland about the size of an almond (larger in the negro than in the Caucasian), is attached by connective tissue to the periosteum at the upper and outer part of the base of the orbit, in a depression known as the lacrimal fossa. Its secretion obtains exit by a number of ducts (six to ten) which open upon the conjunctiva near the outer canthus, above the outer tarsal plate.

The *accessory lacrimal glands* are about forty in number. The largest of these is the one ordinarily described by anatomists. It occupies the loose connective tissue of the eyelid at its temporal extremity, being separated from the orbital lacrimal gland by its capsule, the levator muscle, and Müller's muscle. In many subjects the lobules of this gland invade the lower lid, as well as the upper. It presents several ducts, and is composed of a varying number of lobules. Other accessory glands are found in the connective tissue of the fornix of the conjunctiva, particularly in the temporal half of the lid, although a few are situated near the inner canthus. All these glands present the same lobulated structure. Their ducts are lined with cylindric epithelium. The orifices of the ducts are surrounded by lymphoid infiltration: a condition causing the erroneous

belief that in the normal conjunctiva of man lymph-follicles exist. Waldeyer and Alt have never found true lymph-follicles in the human conjunctiva. The orbital lacrimal gland, the largest of the accessory glands, and probably the smaller ones also, receives branches from the facial and probably from the sympathetic. According to Goldzieher, the secretory nerve of the lacrimal gland is not the fifth, but the facial nerve, whose fibres run through the large superficial petrosal nerve to the spheno-palatine ganglion and thence to the second branch of the fifth nerve.

Fig. 23.—The nasal duct and lacrimal gland. (AUTHOR.)

(Original drawing by DR. R. W. MILLS.)

The lacrimal gland has been pulled forward from its bed.

The excretory part of the lacrimal apparatus begins with the *puncta lacrimalia,* which are minute orifices situated at the inner end of each lid-margin. They mark the beginning of the *canaliculi.* Each canaliculus consists of a vertical part, from 1.5 to 2 millimetres in length, and a horizontal part which measures 7 or 8 millimetres. They end in the lacrimal sac, either by separate orifices or by a conjoint opening. The canaliculi are lined with eight to twelve layers of laminated polygonal pavement epithelium. The *lacrimal sac,* situated in a bony depression at the inner

lower angle of the orbit, is placed behind the tendo oculi and in front of Horner's muscle. Its upper end is rounded; below it shows a marked contraction where it joins the nasal duct. The sac is a slit-like opening composed of fibrous and elastic tissues, lined with a basal layer of cuboid and an inner layer of cylindric cells. In the sac the lining membrane is disposed in numerous folds. When distended the diameter of the sac is 6 or 7 millimetres and its length is 12 millimetres. The *nasal duct* extends from the lacrimal sac downward from 12 to 14 millimetres, but may be prolonged in the nasal mucous membrane and measure 20 or 23 millimetres. It opens beneath the inferior turbinated bone by a vertical or oblique slit. The nasal duct presents many variations in size. Mr. Henry Power, of London, found in 205 European skulls the diameter to average 3.77 millimetres; and in 181 negro skulls, 4.7 millimetres. The direction of

Fig. 24.—Probes introduced into the canaliculi to show their direction. (AUTHOR.)
(Original drawing by DR. R. W. MILLS.)

the canal is downward, backward, and outward. The lining membrane of the duct is continuous with the conjunctiva above and with the nasal mucous membrane below. It is covered with cylindric epithelium. The underlying structure is richly provided with lymphoid tissue. A dense network of veins exists between the mucous membrane and the bony walls of the nasal duct.

The Eyelids.—The base of each orbit is closed by soft structures, viz.: the eyelids. These are movable folds of skin, strengthened by dense, fibrous tissue, and lined internally with mucous membrane. The upper lid is the larger and more movable. At the margin of each lid the skin and mucous membrane pass into each other, forming two lips on the lid-margin. The inner lip is nearly a right angle, the outer is rounded. The orifices of Meibomian glands open in front of the posterior inner lip, while numerous hair-follicles and cilia are found on the rounded anterior lip.

At their free margins the lids are about 2 millimetres in thickness. At the outer and inner angles of the eye the lids are united. The junction points are called canthi. The interval between the canthi varies, averaging about 28 millimetres, and according to its extent gives the appearance of a large or a small eye. Throughout the greater part of their extent the lids are closely applied to the eyeball, but at the inner canthus a vertical fold of conjunctiva, the *plica semilunaris,* intervenes. The plica is the rudiment of the third eyelid, membrana nictitans, found in many animals. Internal to the plica, and filling the inner canthus, is a reddish elevation, the *caruncula lacrimalis,* which is composed of skin containing

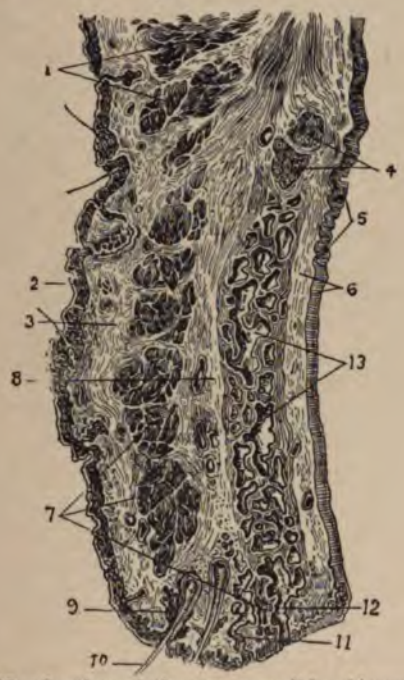


Fig. 25.—Section of the upper eyelid. (AUTHOR.)

1, Orbicularis palpebrarum muscle. *2*, Skin. *3*, Superficial fascia. *4*, Waldeyer's glands. *5*, Papillæ of the conjunctiva. *6*, Conjunctiva. *7*, Orbicularis palpebrarum muscle. *8*, Median connective tissue (tarsal plate). *9*, Sebaceous gland of cilium. *10*, Eyelash. *11*, Gland of Moll. *12*, Duct of Meibomian gland. *13*, Meibomian glands.

modified sweat-glands, sebaceous glands, and hairs. The inner extremity of each lid-margin presents a small elevation, in the centre of which is an orifice, the *punctum lacrimalis,* which is the superior extremity of the channel for the removal of tears. Above and below, the conjunctiva is reflected from the lid on to the globe, the connecting portions being called *the fornices.* The upper fornix is much deeper than the lower.

STRUCTURE OF THE LIDS.—Commencing externally, the skin of the lid is thin and lax, and is covered with fine hairs having sebaceous follicles. There are also sweat-glands. The glands are imbedded in the *subcutaneous*

areolar tissue, which joins the integument loosely to the *orbicularis muscle.* The orbicularis presents palpebral, orbicular, and ectorbital fibres. The palpebral fibres form the sphincter muscle of the eyelids. They arise from the internal palpebral ligament (tendo oculi): a small white cord which passes from the inner canthus to the nasal process of the superior maxilla. The fibres pass backward, traversing the upper and lower lids, and unite at the outer canthus by a cellular raphé, a part being inserted into the external tarsal ligament and malar bone. The orbicularis sends fibres to surrounding muscles. It glides easily over the tarsal plate, but is attached by slips to the integument. A marginal fasciculus of the orbicularis, lying between the eyelashes and posterior lid-margin, has been named the *muscle of Riolan.* On the free margin of each lid is a dark line which runs from one extremity to the other—*the intermarginal line.* It corresponds to the anterior part of the tarsus and marks the union of the cutaneous and tarsal portions of the lid.

Beneath the fibres of the orbicularis is a layer of connective tissue, the *median connective tissue.* Beneath it is found the *tarsal plate,* incorrectly known as the tarsal cartilage, which contains no cartilage cells. The tarsal plate is a firm, flat body composed of fibrous tissue. The upper plate is the larger. They serve to give form to the lids. At the inner canthus the tarsi are fixed to the nasal process of the superior maxillary bone by the tendo oculi, and externally they pass into the external palpebral ligament. The free margin of the tarsal plate is inseparable from the corium of the lid-margin. The orbital margin of each plate passes into the *tarso-orbital fascia,* or septum orbitale. This connects the tarsus to the margin of the orbit. In the upper lid the tarso-orbital fascia blends with the tendon of the levator palpebræ. When the lids are closed the tarsal plates and tarso-orbital fascia form a firm, fibrous covering for the eyeball. Imbedded in the tarsal plates are, in the upper lid, about thirty-five, and, in the lower, about twenty-four *Meibomian glands.* They are long, acinous glands placed nearly parallel to each other. Each gland consists of a vertical duct into which lateral tubular acini open. The opaque fatty matter which they contain permits them to be easily seen through the conjunctiva. Their ducts open upon the lid-margin. The ducts are placed between the conjunctiva behind and the tarsus in front. The Meibomian glands secrete the palpebral smegma, which lubricates the edge of the lid. The mouths of the ducts are lined with stratified epithelium, while the tubes and glandular recesses have a lining of cubical epithelium filled with fatty secretion. *Waldeyer's glands,* known as acino-tubular glands, are composed of acini consisting of a basement membrane lined with cylindric cells placed around a central lumen. They present an oval or round nucleus near their base. Their excretory ducts are lined with cylindric epithelium and end in the palpebral conjunctiva. They have been wrongly called muciparous glands. At the margin of the lid, in front of the muscle of Riolan, are the ducts of the *glands of Moll,* which

are modified sweat-glands resembling the ceruminous glands of the ear. The *eyelashes* are strong hairs arranged in two or three rows along the line of union of the conjunctiva with the skin. Those of the upper lid are the longer. The hairs curve in an opposite direction in the lids, so that their convexities face each other. The life-period of each hair is about four months. They are present in all stages of growth. The posterior part of each lid is the *conjunctiva,* a mucous membrane which covers the lid and is reflected on to the eyeball. The palpebral portion is intimately connected with the tarsal plate, while the ocular portion is loosely attached to the globe. Hence the impossibility of closing up rents in the

Fig. 26.—Arteries of the (right) eyelids. (AUTHOR.)

(Original drawing by DR. H. W. MILLS.)

1, Anastomosis between the lacrimal and superficial temporal. *2*, Superficial temporal. *3*, Supra-orbital. *4*, Fronto-nasal. *5*, Transverse facial. *6*, Superior palpebral. *7*, Infra-orbital. *8*, Inferior palpebral. *9*, Facial.

conjunctiva of the lids, and the ease with which the ocular part of this membrane may be lifted up to close losses. Histologically the conjunctiva consists of stratified columnar epithelium placed upon a connective-tissue matrix. There is in the matrix a certain amount of diffuse adenoid tissue. True lymph-follicles are not found in the human conjunctiva (Waldeyer). The ocular conjunctiva presents the same structure as the palpebral portion except at the corneal margin, where the epithelium changes from the columnar to the stratified squamous type. Lying next to the lid-borders is a strip of conjunctiva, about 3 millimetres broad, which likewise is

covered with stratified squamous epithelium. It is noteworthy that these are the only parts of the conjunctiva free of involvement in trachoma.

Blood-vessels, Lymphatics, and Nerves of the Lids. — The *Arteries* of the eyelids are the internal and external palpebral, the former being derived from the ophthalmic and the latter from the lacrimal. The vessels pass from the outer and inner angles toward the centre of the lid, forming an arch, the *tarsal arch*, along the edge of the lids. A second arch, the *external tarsal arch*, is found in the upper eyelid, where it runs in front of the upper edge of the tarsal plate. The same arrangement is found sometimes in the lower lid. The tarsal arches are joined by small anastomosing branches.

Fig. 27.—Veins of the (left) eyelids. (After Soemmering.)

1, Branch to the deep temporal vein. *2*, Facial vein. *3*, Supra-orbital. *4*, Angular. *5*, Branch connecting the temporal and facial. *6*, Dorsal vein of the nose. *7*, Frontal vein.

The *Veins* of the eyelids are disposed in two series: the pretarsal and the post-tarsal. The former empty into the superficial temporal and facial veins, while the latter pass into the ophthalmic vein.

There are likewise two networks of *Lymphatics*, which follow the corresponding veins. The networks are connected by vessels which pierce the tarsi. The lymphatics empty into the submaxillary, pre-auricular, and parotid lymphatic glands. The pre-auricular gland is often enlarged in gonorrheal ophthalmia.

The *Nerves* of the eyelids are as follows: The levator palpebræ is supplied by the third and the orbicularis palpebrarum by the facial. The sensory nerves are branches of the fifth.

ANATOMY OF THE EYEBALL.

The eyeball is a spheroid, its average measurements in the adult being 24.3 millimetres in its antero-posterior diameter, 23.6 millimetres transversely, and 23.4 millimetres vertically. It presents in front a transparent segment of a sphere, the cornea, which forms about one-sixth; and posteriorly the opaque sclerotic, forming about five-sixths of the globe. The cornea and sclera form the outer, or fibrous, coat of the eye. The second coat consists of the iris, ciliary body, and chorioid, which are vascular and pigmentary structures. The third, or innermost, coat is the retina, which is the expansion of the optic nerve. The nerve enters the globe 3 millimetres internal to the posterior pole. The average weight of the eye is 7.2 grammes; its volume equals about 6 cubic centimetres. At birth its weight is 2.2 grammes; and its antero-posterior diameter measures 15.4 millimetres. Its greatest growth occurs during the first year (Weiss).

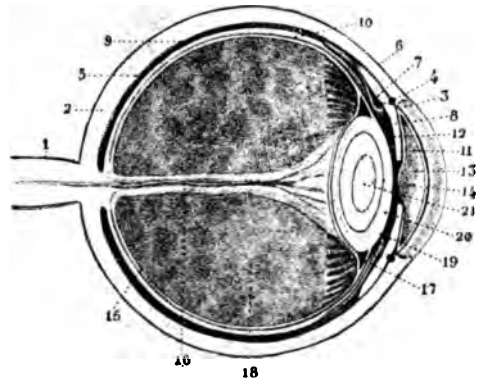


Fig. 28.—Antero-posterior section of the eyeball. (LÉVEILLÉ.)

1, Optic nerve. *2*, Sclerotic. *3*, Cornea. *4*, Spaces of Fontana. *5*, Chorioid. *6*, Ciliary muscle. *7*, Ciliary processes. *8*, Iris. *9*, Retina. *10*, Jacob's membrane. *11*, Anterior chamber. *12*, Posterior chamber. *13*, Pupillary area. *14*, Aqueous humor. *15*, Hyaloid membrane. *16*, Canal of Stilling. *17*, Canal of Petit. *18*, Vitreous humor. *19*, Capsule of the lens. *20*, Fluid of Morgagni. *21*, Lens.

The anterior and posterior extremities of a sagittal section are called the poles of the eye, while a plane dividing the globe into a fore and hind half is called the equator. In an adult the equatorial circumference averages 77.6 millimetres. Within the globe are contained transparent media: the vitreous behind, the aqueous in front, and the crystalline lens intermediately. We must now consider these structures *seriatim*.

The Fibrous Coat.—CORNEA.—This transparent structure, freely supplied with nerves, but devoid of blood-vessels, is fitted into the sclera like a watch-glass. It measures 11 millimetres in the vertical, and 12 millimetres in the horizontal, diameter. It is thickest at the rim (1.2 millimetres), and at its centre measures 0.8 millimetre. The anterior surface of the cornea is convex; the posterior surface is concave, and is of greater extent than the anterior. The sclera overlaps the cornea, forming a

whitish ring called the *limbus*. Around the corneoscleral junction is a slight groove, the *sulcus scleræ*. The cornea consists of five layers. The outermost of these is the *epithelium*, which is continuous with the epithelium of the conjunctiva, and consists of six or eight layers of cells at its centre and more at the periphery. The superficial cells are flat, the central ones irregularly polygonal prickle-cells, and the basal layer is columnar. The next layer, the *anterior elastic lamina*, or *Bowman's layer*, is a thin, homogeneous lamina, which is thickest in the middle and tapers toward the periphery. It measures 0.01 to 0.02 millimetre in thickness. Although it presents no corpuscles, this layer does not differ materially from the next layer, the *true corneal tissue*, or *substantia propria*. This comprises the greater part of the cornea, is continuous with the sclera, and consists of fibrillæ which form about twenty bundles (lamellæ). The fibrillæ are cemented together, and, while running parallel with the gen-

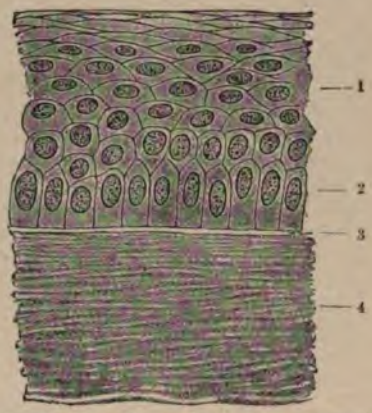


Fig. 29.—Section through the human cornea.
(BÖHM and DAVIDOFF.)

1, Anterior epithelium. *2*, Basal cells. *3*, Bowman's layer. *4*, Substantia propria. × 500.

eral surface of the cornea, they intersect in various ways. It is in this layer that the *corneal spaces* are found. Between the lamellæ are irregular stellate cell-spaces, which can be demonstrated by staining the tissue with nitrate of silver or chlorid of gold. Passing from the spaces, or *lacunæ*, are numerous *canaliculi*, which connect each cell-space with other lacunæ, constituting a system of lymph-passages. It is by means of the circulation of lymph that the cornea is nourished. These spaces interlace up to the corneal border, where they anastomose with the lymphatic vessels of the conjunctiva. Contained within the cell-spaces are the *corneal corpuscles*, which are distinguished as *"fixed"* and *"wandering."* The fixed corneal corpuscles are anchored to the wall of the lacuna. They do not completely fill the lacuna, and from the body of the cells protoplasmic processes pass in every direction into the canaliculi, thus connecting with other corpuscles. Since the corpuscles and their processes do not fill the spaces

in which they lie, room is obtained for the circulation of lymph. The *wandering cells* are lymph-cells which have found their way into the cornea. In the normal cornea they are few in number; but under pathologic conditions they are numerous, escaping from the marginal network of blood-vessels. The fourth layer is known as the *posterior elastic lamina: membrane of Demours* or *of Descemet.* This is a homogeneous, elastic layer which forms the posterior boundary of the cornea, and, unlike the anterior elastic lamina, is sharply separated from the true corneal layer. It resists acids and alkalies, but is digested by trypsin. It possesses great resisting powers to pathologic processes (and it often prevents perforation of a corneal ulcer). In thickness it varies from 0.006 to 0.012 millimetre, the thinnest portion being central. At its periphery it breaks up into bundles of fibres, to some of which the ciliary muscle is attached. A few fibres extend around the angle of the anterior chamber and pass into the substance of the iris. They form what is called the *ligamentum pectinatum.*

Fig. 30.—Corneal corpuscles of the dog.
(BÖHM and DAVIDOFF.)

These fibres are more numerous in the eyes of some lower animals (the sheep and ox) than in man. Between the fibres passing from the cornea to the iris are the *spaces of Fontana,* which are not well marked in man. The *endothelium,* or fifth layer of the cornea, consists of a single layer of flattened six-sided cells which line the posterior surface of Descemet's membrane.

Blood-vessels of the Cornea.—In the adult vertebrates the centre of the cornea has no blood-vessels. In man vessels in the form of loops are found at the periphery of the cornea, forming a zone from 1 to 1.5 millimetres broad. These arise from the superficial conjunctival arteries.

Lymphatics have not been found in the cornea.

Nerves.—The nerves of the cornea are numerous and are derived from the long ciliary nerves via the nasal branch of the ophthalmic division of the fifth, the short ciliary nerves of the ciliary ganglion, and a few conjunctival branches (lacrimal and infratrochlear nerves). These pass into

the cornea. At a distance of 1 to 2 millimetres from the limbus they lose the medullary covering, the axis-cylinders forming a plexus, the *fundamental plexus,* in the anterior part of the substantia propria. From this plexus branches pass through Bowman's membrane, forming a *subepithelial plexus,* from which small varicose fibrils pass among the epithelial cells. The primary plexus also gives off branches to the posterior layers of the cornea. Some of the finest nerve-fibrils run in the lacunæ and canaliculi, where they are in intimate relationship with the corneal corpuscles. A connection between these nerve-filaments and the corpuscles has not been demonstrated.

SCLERA.—The sclerotic forms the posterior five-sixths of the outer, or fibrous, coat of the eye. It is a white, opaque membrane, which gives shape to the eye and receives the insertions of the ocular muscles. At its

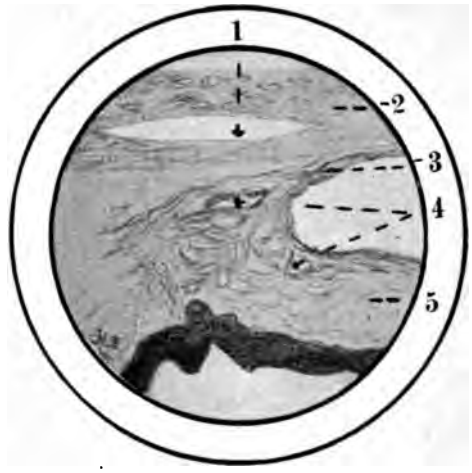


Fig. 31.—The iridocorneal angle. (AUTHOR.)

1, Canal of Schlemm. *2*, Cornea. *3*, Ligamentum pectinatum. *4*, Spaces of Fontana. *5*, Iris.

posterior part it is pierced by the optic nerve, and here the sclera is thickest (1.1 millimetres). From this point it becomes thin as it is continued forward, and is only 0.3 millimetre thick in the anterior part under the recti muscles. It becomes thicker (0.6 millimetre) at the insertion of the recti tendons. It is composed of white, fibrous tissue, elastic fibres, connective-tissue corpuscles, and pigment-cells. The bundles of white, fibrous tissue cross at right angles, some being continuations of recti tendons, others following the course of the obliques. The sclera contains few blood-vessels, no true lymphatic vessels, and a few nerves derived from the ciliary (short and long) nerves. Posteriorly, about 3 millimetres internal to the antero-posterior axis, the optic nerve pierces the sclera, the axis-cylinders entering the eye through the *lamina cribrosa*. This part will be more fully described in connection with the optic nerve. Externally the sclera is smooth, and is connected with the conjunctiva by loose con-

nective tissue, the *episcleral tissue.* Internally the sclera presents a number of fine grooves for lodgment of the ciliary nerves, and a delicate membrane, the *lamina fusca,* which belongs to the next coat of the eye. Anteriorly the sclera is continuous with the cornea, and here the sclera presents a beveled edge. The line of union is more peripherally situated internally than externally. Hence the sclera overlaps the external layers of the cornea.

The Vascular Coat.—The vascular and pigmentary coat consists of the iris, ciliary body, and chorioid, collectively called the uveal tract. The iris forms a curtain, or movable diaphragm, placed in front of the lens and suspended in the aqueous humor. It separates the anterior from the posterior chamber. The opening in the iris is called the pupil. It is almost circular, and is placed slightly to the inner side of the centre of the iris. It constantly varies in size during life, and its average diameter

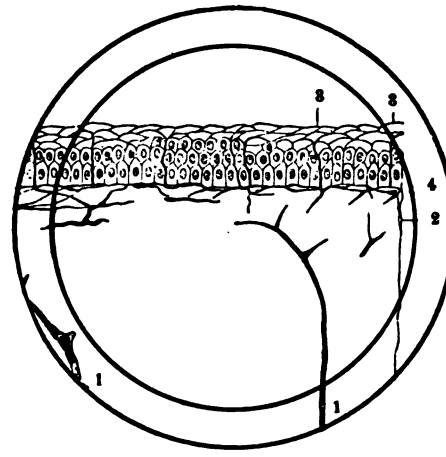


Fig. 32.—Corneal nerves of the pig. (After ROLLETT.)

1, 1, Larger nerves. *2,* Plexus beneath Bowman's layer. *3, 3,* Terminal twigs ascending through the epithelium. *4,* Subepithelial plexus.

is 4 millimetres. The color of the iris varies with the age of the subject and the latitude of the country. In the newborn the iris is of a light grayish-blue color; later it becomes darker from development of the stromal pigment. In northern countries blue and gray irides are the most frequent.

STRUCTURE OF THE IRIS.—The framework of the iris is connective tissue, inclosing blood-vessels, nerves, pigment-cells, and muscular fibres. The anterior surface, except at the pupillary edge, is uneven, and presents crypts and folds. The latter are of two kinds: structural, or permanent, folds; and contraction folds, which vary. The *anterior layer* of the iris is an epithelial covering continuous with the fifth layer of the cornea. In dark-skinned races these cells contain pigment-granules. *The stroma* of the iris consists of cells and fibres of connective tissue forming a delicate mesh in which vessels and nerves are found, as well as pigment-granules. The *muscular tissue* is of the involuntary variety and presents two kinds

of fibres: circular and radiating. The circular fibres are on the posterior surface of the iris and surround the pupil, forming a circular zone 0.6 millimetre broad, called the *sphincter pupillæ.* The radial muscular fibres —*dilator pupillæ*—pass from the periphery toward the pupil and mingle with the circular fibres. The *pigment* varies in situation in various irides. In light-colored eyes the only pigment is that found on the posterior surface of the iris. This layer is absent in albinos. In dark eyes pigment-granules are found in the stroma of the iris and in the cells of the anterior surface in addition to the posterior layer. The *posterior layer,* called the retinal part of the iris, is made up of pigment-cells and is a continuation of the pigmentary layer of the retina, which consists of two layers of pigment. These pass forward in a wavy course to the pupillary edge of the iris, where they unite. This layer turns slightly forward, as can be seen in Fig. 33.

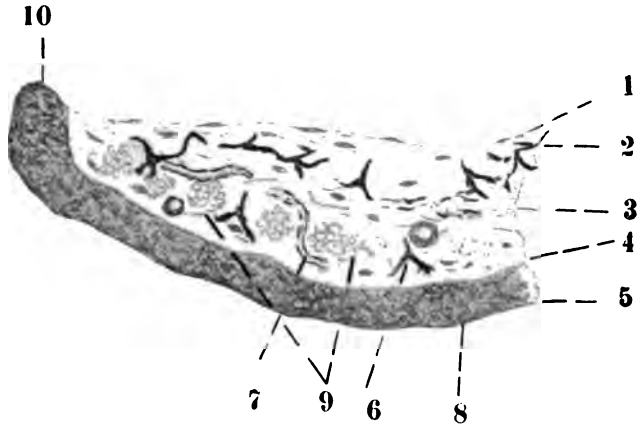


Fig. 33.—Radial section of the human iris. (AUTHOR.)

(Original drawing by DR. CARL FISCH.)

1, Endothelial layer. *2*, Anterior limiting layer. *3*, Vascular layer. *4*, Posterior limiting layer. *5*, Pigment layer. *6*, Pigment-cell. *7*, Capillary. *8*, Internal limiting membrane. *9*, Sphincter pupillæ muscle. *10*, Pupillary margin.

The Arteries of the Iris are branches from the long and anterior ciliary and the vessels of the ciliary processes. The two long ciliary arteries pierce the sclera on each side of the optic nerve, pass forward between the sclera and chorioid, and divide into branches forming a vascular ring, *large arterial circle,* around the periphery of the iris. From this circle small branches pass to the ciliary muscle, while others run toward the pupil, around which they form the *small arterial circle.* The five or six anterior ciliary arteries arise from the muscular and lacrimal branches of the ophthalmic artery and pierce the sclera just behind the cornea. They supply the ciliary processes and join the great arterial circle. In addition to these, other small arteries pass from the ciliary processes to the iris. The anterior conjunctival arteries are small vessels which become prominent in inflammatory conditions of the conjunctiva.

The Veins of the Iris follow the course of the arteries, and end in the venæ vorticosæ.

The Nerves of the Iris are numerous. They come from the ciliary branches of the lenticular ganglion and the long ciliary. There are about fifteen of the former. They pierce the sclera around the optic nerve, pass forward imbedded in grooves on the inner surface of the sclera, and communicate before distribution. They supply the cornea, ciliary muscle, and

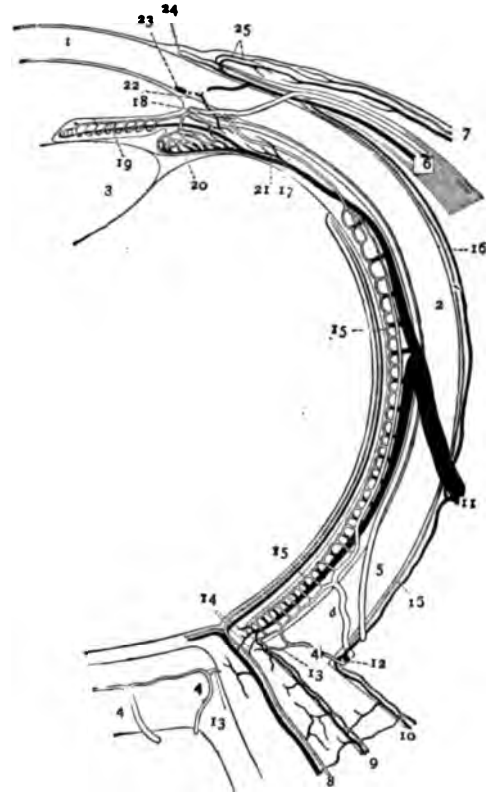


Fig. 34.—Diagram of the vessels of the eye. (LEBER.)

1, Cornea. *2*, Sclera. *3*, Lens. *4*, *4*, Short posterior ciliary arteries. *5*, Long posterior ciliary artery. *6*, Anterior ciliary artery and vein. *7*, Posterior conjunctival artery and vein. *8*, Central retinal artery and vein. *9*, Vessels of the internal optic sheath. *10*, Vessels of the external optic sheath. *11*, Vena vorticosa. *12*, Posterior short ciliary vein. *13*, Branch of short posterior ciliary artery to the optic nerve. *14*, Anastomosis of chorioidal vessels with those of optic nerve. *15*, Chorio-capillaris. *16*, Episcleral branches. *17*, Recurrent chorioidal artery. *18*, Large arterial circle of the iris (transverse section). *19*, Vessels of the iris. *20*, Ciliary process. *21*, Branch of vena vorticosa from the ciliary muscle. *22*, Branch of anterior ciliary vein from the ciliary muscle. *23*, Canal of Schlemm. *24*, Plexus of the corneal margin. *25*, Anterior conjunctival artery and vein.

iris. In the iris they follow the course of the vessels and form a plexus of non-medullated fibres. Fibres from the motor oculi supply the sphincter pupillæ. The nerve-supply of the dilator fibres is in dispute. Many authors state that the dilator is supplied by the sympathetic, but late researches show that the dilator fibres do not have their course exclusively

through the cervical sympathetic. After excision of the superior cervical ganglion and an inch of the sympathetic nerve below it, the author has found the pupil reflex to be present. This observation seems to confirm the view of those physiologists who hold that dilating fibres pass out directly from the brain along the fifth nerve.

THE CILIARY BODY includes that portion of the uveal tract placed between the termination of the chorio-capillaris, opposite the ora serrata behind, and the periphery of the iris in front. It is connected with the lens by the zonula of Zinn. The ciliary body is in the shape of a triangular prism with its acute angle placed posteriorly and bent upon itself so as to form a ring. The uveal tract is closely attached to the fibrous coat at

Fig. 35.—Injected blood-vessels of iris, ciliary body, and chorioid of man. × 7. (BÖHM and DAVIDOFF.)

only two places: one is around the optic-nerve entrance; the other is at the point where the tendon of the ciliary muscle is attached to corneo-scleral tissue. The ciliary body consists of the ciliary muscle, ciliary processes, ciliary glands, and the usual connective tissue, blood-vessels, and nerves. To examine these parts *in situ* the student should cut away the posterior three-fifths of the eye. Looking from within, this part of the uveal tract is seen to consist of three parts: *the ciliary ring, the ciliary processes,* and *the ciliary muscle.* The ciliary muscle, however, cannot be seen from behind; it must be examined in an antero-posterior section of the globe. *The ciliary ring,* or *orbiculus ciliaris,* is a circular tract, 4 millimetres broad, extending from the ora serrata to the ciliary processes.

In this zone we note, in contradistinction to the chorioid, the absence of chorio-capillaris, the presence of muscular fibres connected with the ciliary muscle, and the change in the stroma, which here consists of fibrous connective bundles instead of elastic layers.

The Ciliary Processes, the most internal part of the uveal tract, have the same structural arrangement as the chorioid, being composed of pigmented epithelial cells, non-pigmented cylindric cells, homogeneous intercellular substance, blood-vessels, and nerves. In the ciliary processes, unlike the chorioid, there are no capillaries, the arteries passing directly

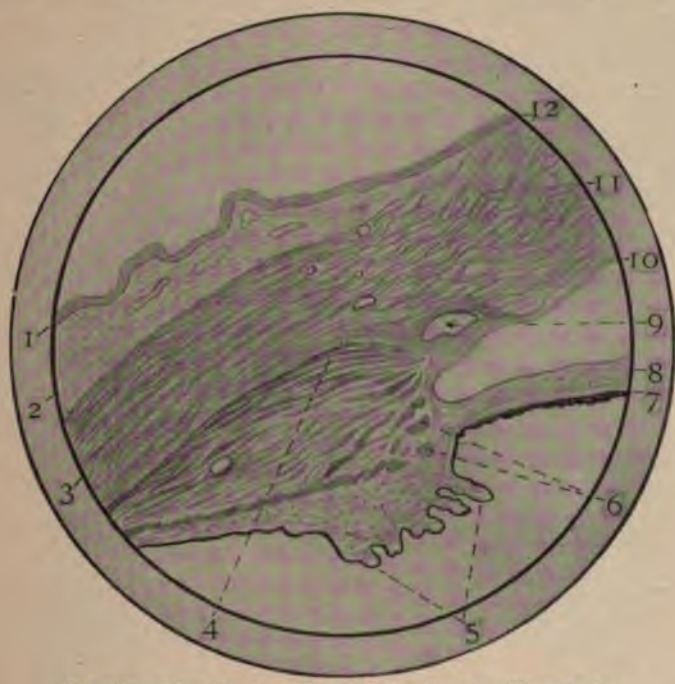


Fig. 36.—Meridional section of the human ciliary body.
(After Böhm and Davidoff.)

1, 2, Conjunctiva. *3,* Sclera. *4,* Meridional fibres of the ciliary muscle. *5,* Ciliary processes. *6,* Circular fibres of the ciliary muscle. *7,* Iris pigment. *8,* Stroma of the iris. *9,* Canal of Schlemm. *10,* Membrane of Descemet. *11,* Cornea. *12,* Corneal epithelium.

into the veins. The processes present glandular invaginations of the pigmented epithelium covering their free surface. These will be described elsewhere as the ciliary glands. The ciliary processes are seventy to eighty in number. They form the radial vascular folds projecting from the inner surface of the ciliary body. After rising to the height of 1 millimetre they end at the base of the iris.

The Ciliary Muscle forms the third zone, or division, of the ciliary body. It consists of *radial and circular fibres.* The former arise from the internal surface of the corneoscleral junction, between the canal of Schlemm and the anterior chamber. They pass backward to the ciliary

processes and orbiculus, and constitute the radial ciliary muscle. These fibres are attached to the chorioid coat opposite the ciliary processes and farther back. Hence the name *tensor chorioidæ* given by Brücke. The size of this muscle varies in different eyes according to the antero-posterior diameter. Thus, in the short (hypermetropic) eye the muscle is smaller than in the long (myopic) eye, as can be seen in Figs. 38 and 39. Many of the shorter radial fibres pass out of their meridional course and join the circular fibres, or Müller's annular muscle. This part of the ciliary muscle is placed internal to the longitudinal fibres, and forms a circular ring around the periphery of the iris at the base of the ciliary processes.

Ciliary Glands.—In the ciliary body, in front of the ora serrata, are small processes, each consisting of a group of cells (Fig. 40). The cells are arranged in rings with a central lumen. These glands, according to Nicati and Collins, secrete the aqueous humor, and are the seat of pathologic changes in serous iridocyclitis. It is proper to state, however, that

Fig. 37.—Meridional section of human ciliary processes. (PIERSOL.)

I, Interstitial connective-tissue stroma, covered by retinal layers (*R*). *i*, *o*, Inner clear and outer pigmented layers of cells. *f*, Fibrous tissue of the processes.

some observers do not accept this description of the ciliary glands, although the researches of Treacher Collins seem to have established their existence.

The Blood-vessels and Nerves of this part of the eye have been described in connection with the iris.

THE CHORIOID.—The chorioid is a dark-brown membrane placed between the sclera and retina. It is separated from the sclera by a large lymph-space traversed by a loose meshwork of fine connective fibres forming the suprachorioid membrane. In this, stellate pigment-cells are found, irregularly disposed or in patches; and lymphoid cells also occur here. The *lymph-space* between the chorioid and sclera is lined by endothelial cells, and communicates with the capsule of Tenon at the points where the vessels and nerves pierce the sclera. The chorioid proper consists of a connective-tissue stroma, supporting blood-vessels of different sizes. The following layers are noted from without inward: The *layer of large vessels;* the *layer of small vessels,* or *chorio-capillaris;* and the *homogeneous layer,*

or *lamina vitrea*. The *layer of large vessels*, also called *tunica vasculosa Halleri*, is composed chiefly of veins closely arranged and often anastomosing. The intervascular spaces contain numerous pigment-cells. A surface view of this layer, examined in the living eye with the ophthalmoscope, shows a plexus of bright lines, the vessels resting upon a dark background. This is the picture to which the name of tessellated fundus is given. The largest vessels of this layer are placed superficially (externally); the smallest are most internally placed, adjacent to the chorio-capillaris; and the vessels of medium size occupy a midway position. Of the largest blood-channels, the four *venæ vorticosæ* are most prominent. Their tributaries collect

Fig. 38.—Section through the ciliary region of an hypermetropic eye. (IWANOFF.)

Fig. 39.—Section through the ciliary region of a myopic eye. (IWANOFF.)

blood from the chorioid, ciliary body, and iris. The veins of the chorioid are inclosed in lymph-spaces: *perivascular lymph-sheaths*. The capillaries of this layer are derived from the terminal branches of the short ciliary arteries. The *chorio-capillaris*, misnamed *membrana Ruyschii*, consists of large-sized capillaries closely placed with very narrow interspaces and no pigment. The *lamina vitrea* is the most internal layer of the chorioid. It supports the pigment-layer of the retina.

The Tapetum.—In some animals the stroma between the large vessels and the chorio-capillaris is fibrous, and, being highly reflective, causes their eyes to shine. This layer is called the tapetum. In many of the

domestic animals it consists of wavy bundles of connective tissue. In the carnivora the reflex is due to the presence of plate-like cells containing innumerable small crystals.

Fig. 40.—Bleached section showing glands of the ciliary body. × 300. (TREACHER COLLINS.)

ANATOMY OF THE CORNEOSCLERAL JUNCTION.—The fourth layer of the cornea, near the corneoscleral junction, divides into a number of fibres. Some of these give attachment to the ciliary muscle, while others pass around the angle of the anterior chamber to join the iris and form the

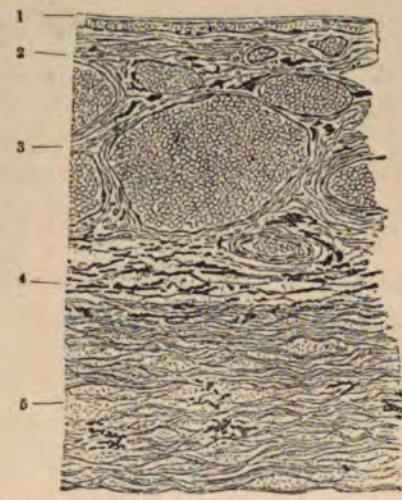


Fig. 41.—Vertical section of the chorioid. (BÖHM and DAVIDOFF.)

1, Lamina vitrea. *2*, Chorio-capillaris. *3*, Layer of large vessels. *4*, Suprachorioidea. *5*, Sclera. × 130.

ligamentum pectinatum iridis. The fibres passing to the iris are covered with endothelial cells continued from the membrane of Descemet; but these cells do not cover the interspaces between the bundles of fibres, and for this reason the fluid in the anterior chamber communicates freely with

the interspaces. The spaces in this sponge-like tissue are known as the *spaces of Fontana,* and are larger in the eyes of the ox and sheep than in the human eye. A similar, but larger space, the *canal of Schlemm,* is found within the sclera at its junction with the cornea. It is a flattened circular channel, elliptic in section, and is often double or treble in parts of its course. It communicates with Fontana's spaces, and by these with the aqueous chamber. It also is connected with the veins in the anterior part of the sclera. Hence, the aqueous humor can leave the eyeball via the spaces of Fontana, the canal of Schlemm, and the scleral veins. Schwalbe found that spaces and veins were filled with a colored fluid which had been injected into the anterior chamber. The canal of Schlemm must be regarded as a venous channel, although many anatomists believe that it is a lymph-channel. It certainly is the drainage apparatus of the eye. The canal of Schlemm is visible in living eyes possessing a thin sclera (found in young girls). Under oblique illumination it appears as a dark line concentric with the corneal margin. The dark color indicates that the canal in life contains blood (Fuchs).

IRIDOCORNEAL ANGLE. — The point of junction of the iris, cornea, sclera, and ciliary muscle forms an important part of the eye, both anatomically and pathologically. The angle of the anterior chamber, as this part is sometimes called, becomes blocked in glaucoma.

THE OPTIC NERVE, RETINA, LENS, AND VITREOUS.

The Optic Nerve.—This nerve, derived from the primary optic vesicle of the embryo, is regarded as a part of the brain. It is divisible into cranial, orbital, and ocular portions. The nerve is about 5 centimetres long, of which 3 centimetres are in the orbit, 1 centimetre is in the optic canal, and 1 centimetre is intracranial. Its width is 5 millimetres. The nerve ends anteriorly in the retina. The nerves of opposite sides are connected by the optic commissure. Behind the commissure they are named the optic tracts. Each optic tract has two origins: one from the stratum opticum of the corpora quadrigemina, the other from the optic thalamus. Passing backward the optic tract ends in ganglion-cells of the pulvinar, anterior quadrigeminal, and external geniculate bodies. From these ganglion-cells fibres, named optic radiations, pass backward to end in ganglion-cells of the cortex of the posterior part of the occipital lobes. The optic fibres are distributed to the occipital lobe, including its mesial surface, the cuneus. The cuneus is bounded by the parieto-occipital fissure above and in front, and by the calcarine fissure below. It is this region, the cuneus and the parts around the calcarine fissure, which is the visual area of the cerebrum. Each optic tract winds around the corresponding crus cerebri and passes inward and forward to join its fellow in forming the commissure.

The optic tract and commissure are composed of medullated nerve-fibres

bound together by neuroglia. They have no neurilemma. Their blood-supply comes from small vessels supplying the front part of the brain and pia mater. The optic nerves are composed of medullary fibres, neuroglia, and blood-vessels surrounded by pia mater. The orbital portion of the optic nerve is covered in addition by the arachnoid and dura mater. Lymph-spaces exist in the nerve and between its coverings in the orbit.

The Retina.—The expansion of the optic nerve within the eye is a delicate membrane called the retina. External to it is the chorioid, and internally it rests on the hyaloid membrane of the vitreous body. The retina extends from the entrance of the optic nerve to the pupillary margin of the iris, and is divisible into three zones: (1) the optic part, reaching forward to an irregular, serrated margin situated just behind the ciliary processes (ora serrata); (2) the ciliary part, extending from the ora serrata to the ciliary margin of the iris; (3) the iridal part, covering the posterior surface of the iris. The optic part, alone sensitive to light, is

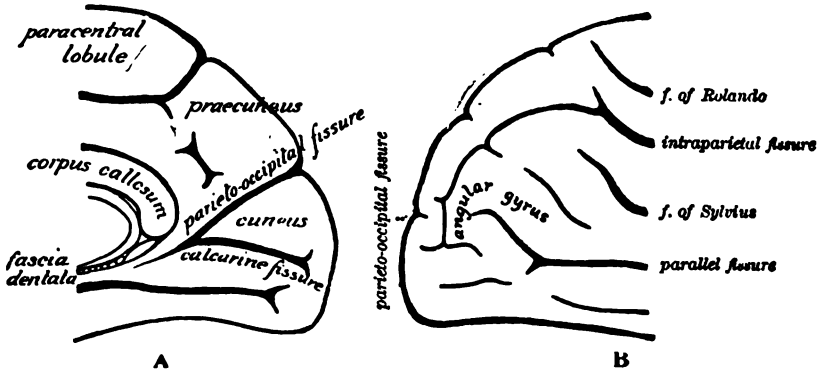


Fig. 42.—Diagram of occipital region of right cerebral hemispheres.
A, from inner, *B*, from outer, aspect.

divisible into: (1) an outer, or neuroepithelial, layer; and (2) an inner, or cerebral, layer. Each of these is microscopically separable into several layers.

The ciliary and iridal parts of the retina contain no nerve-elements, and are simply pigment layers. The retina varies in thickness from the macula (0.5 millimetre) to the ora serrata (0.1 millimetre). Its inner surface is smooth; its outer surface is adherent to the chorioid. The inner surface presents three points of interest: the macula lutea, the fovea centralis, and the porus opticus. The first, known as the *yellow spot,* is in the axis of the globe. It is elliptic in shape, and measures from 1 to 2 millimetres in diameter. In its centre is a depression, the *fovea centralis,* 0.2 to 0.4 millimetre in diameter. Here the structure is much changed, as will be explained later. About 3 millimetres to the nasal side, and 1 millimetre below the yellow spot, is the *porus opticus,* the place where the optic nerve pierces the retina and expands into its inner layer. The optic

disc forms a slight elevation, *colliculus nervi optici*. In its centre is a physiologic excavation, the point from which the retinal vessels branch. The color of the retina is a light pink in the fresh eye; if kept in a dark place for some minutes before removal, it is a purple-red. Exposure to sunlight bleaches it. Boll discovered the retinal purple which Kühne named rhodopsin. It is found in all parts of the retina except the macula, fovea, and ora serrata. After death the retina becomes opaque.

Layers of the Retina.—Microscopic study of the retina has claimed the attention of a host of observers from the time of Heinrich Müller to the present day. Recent researches have resulted in a classification of the retinal layers much different from that of the early writers. The later anatomists, particularly Ramón y Cajal, using the Golgi stain, give the following layers:—

1. Pigment epithelium layer.
2. Layer of rods and cones.
3. Granules of the nerve-cells.
4. Outer plexiform layer.
5. Layer of horizontal cells.
6. Layer of bipolar cells.
7. Layer of amakrine cells.
8. Inner plexiform layer.
9. Ganglion-cell layer.
10. Layer of nerve-fibres.

Fig. 43.—The macula near the fovea centralis. × 80.
(Photomicrograph by Dr. G. A. Dixon of a section by Dr. J. E. Weeks.)

These are shown diagrammatically in Fig. 45, after Golgi's method.

For the purpose of histologic description, the following designations are most conveniently used:—

I. Neuron
1. Pigment epithelium.
2. Layer of rods and cones.
3. Membrana limitans externa.
4. Outer nuclear layer and layer of Henle's fibres.

II. Neuron
5. Outer plexiform layer.
6. Inner nuclear layer.
7. Inner plexiform layer.

III. Neuron
8. Layer of ganglion cells.
9. Layer of nerve-fibres.
10. Membrana limitans interna.

Optic Part of the Retina.—As already mentioned, this part of the retina is divided into two layers, the neuroepithelial and the cerebral, each of which consists of a number of laminæ; so that, including the pigmentary layer, this part is composed of ten different strata. Besides the

nervous epithelial elements, the retina contains supporting structures (not of connective-tissue origin), the most important of which are the radial, or Müller, fibres. They run from the inner surface to the layer of rods and cones. Their distal end is conically enlarged, and the bases of these cones join together to form the so-called membrana limitans interna. Their nuclei lie within the inner granular layer; along their whole course they send out processes in all directions. There are in the outer reticular layer other supportive cells, which are called concentric cells; near the entrance of the optic nerve glia-cells are found. To these supportive structures, too, belong fibres which arise from the membrana limitans externa, and basket-like surround the bases of the rods and cones.

Fig. 44.—Hexagonal cells from the pigment layer of the retina of a rabbit. (AUTHOR.)

(Photomicrograph by DR. H. P. WELLS.)

CEREBRAL LAYER.—The layer of nerve-fibres consists of non-medullated axis-cylinders arranged in bundles. They are most numerous at the site of entrance of the optic nerve, and run radially to the ora serrata (see macula lutea). For the most part they are centripetal fibres derived from the ganglionic layer, but there are centrifugal fibres coming from cerebral ganglionic cells, which end free in this layer.

The layer of ganglion-cells (ganglion nervi optici) is a single layer of large multipolar cells, sending an undivided process (axis-cylinder) to the layer of nerve-fibres and dendritic processes toward the inner reticular layer, where they form a dense network with the corresponding processes of other ganglion-cells.

This inner reticular (plexiform) layer is formed by a very fine network of supportive tissue, in which processes of all the ganglion-cells of the retina are imbedded.

The elements of the inner granular layer are of very different types. Its innermost layer is formed by large multipolar ganglion-cells, which send processes into the inner plexiform layer. Many of them emit also

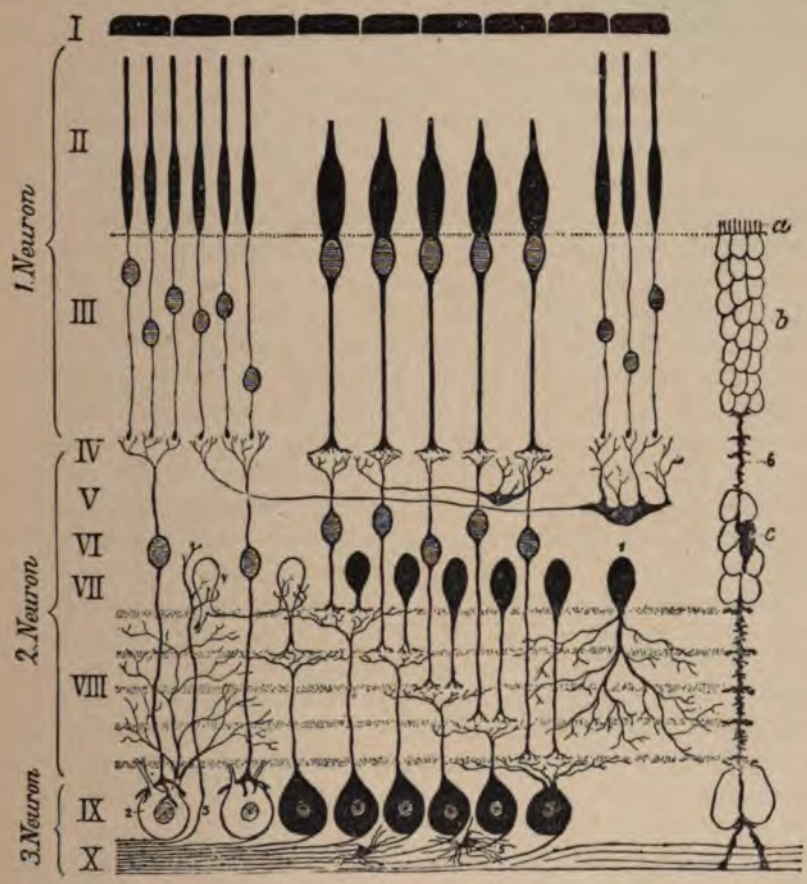


Fig. 45.—Diagram of the structure of the human retina according to Golgi's method. (GREEFF.)

I, Pigment epithelium layer. *II*, Rods and cones. *III*, Granules of the visual cells. *IV*, Outer plexiform layer. *V*, Layer of horizontal cells. *VI*, Layer of bipolar cells. *VII*, Layer of amakrine cells. *VIII*, Inner plexiform layer. *IX*, Ganglion-cell layer. *X*, Layer of nerve-fibres. *1*, Diffuse amakrine cell. *2*, Diffuse ganglion-cell. *3*, Centrifugal nerve-fibre. *4*, Amakrine association fibres. *5*, Neuroglia-cells. *6*, Müller's radial fibres.

an axis-cylinder to the layer of nerve-fibres. The remaining strata of this layer are mostly characterized by the presence of small bipolar cells (ganglion retinæ) which show a centrifugal process ramifying in the inner plexiform layer and another one reaching to the outer reticular layer, where it divides in a maze of fine fibrillæ. All of the bipolar cells are

provided with a process that penetrates between the visual cells and ends with a little thickening at the level of the membrana limitans externa. The most external part of the layer is taken up by large and small spider-shaped cells, from each of which an axis-cylinder is sent to the layer of nerve-fibres, while numerous fine processes enter into the outer plexiform layer.

The outer plexiform layer, like the inner layer, is a network of fibres of the supporting substance, which contains the process just mentioned. In it, too, the concentric cells are found, as well as some displaced ganglion-cells, which belong to the ganglion retinæ.

NEUROEPITHELIAL LAYER.—This lamina contains the rod- and the cone- cells, which are peculiar by the fact that their nuclei lie in the lower half of the cells, while the rest of the cell perforates the membrana limitans externa, thus seemingly appearing separated from the lower half. This peculiarity gives rise to the appearance of two layers, the outer granular layer (the nuclei of these cells) and the layer of the rods and cones. The rods and cones consist of two halves, an outer homogeneous part (cylindric in the rods and conic in the cones), and an inner granular part. In the cones the inner part is thick and swollen. The nuclei of the rod-cells show two or three transverse striæ, and the inner parts of rods and cones contain a peculiar filamentous apparatus. The number of rods exceeds that of the cones considerably. They are arranged in regular intervals. Those portions of these visual cells which are in contact with the outer plexiform layer show a characteristic striation (the fibrous layer of Henle).

The pigmentary lamella finally is formed by a single layer of hexagonal pigmented cells. Their sides toward the chorioidea are pigment-free and contain the nuclei.

This arrangement of the retinal layers is somewhat changed within the macula lutea and the fovea centralis. Delicate optic fibres proceed directly from the entrance of the optic nerve to the nearest medial portion of the macula, while all the shorter fibres pursue a convex course and unite at the periphery of the macula. At the macula the layer of ganglion-cells consists, instead of a single one, of eight to nine layers of cells. The inner granular layer, too, is considerably increased in thickness. The neuroepithelium consists only of cone-cells; so that here the latter alone form the layer of Henle.

Toward the fovea the layers of the retina become gradually thin. The layer of nerve-fibres almost disappears and the layers of the cerebral portion confluesce to a thin lamina; so that in the centre the fovea consists only of the neuroepithelium.

Nearing the ora serrata one after another the different layers become thinner and finally disappear; only the radial fibres persist.

CILIARY AND IRIDAL PARTS OF THE RETINA.—The former consists of a single layer of elongated cylindric cells, which originates from the union between the outer and inner granular layer. Their surface is covered by a cuticula, while on the outside they are connected with the pigment layer.

In the iridal part we distinguish two layers: an anterior, with spindle-shaped, and a posterior, with polygonal, pigment-cells. The posterior surface is covered with a thin cuticula, the limitans iridis, which is derived from the membrana limitans interna retinæ.

As to the connection of the nervous elements of the retina, the cells of the ganglion nervi optici, as well as the star-shaped cells of the inner nuclear layer, furnish the centripetal optic fibres, while the centrifugal fibres end free in the inner nuclear layer. The cells of the ganglion retinæ do not possess axis-cylinders. Their connection with the rest of the nervous elements is effected by means of the nervous plexus in the inner and outer plexiform layer. The visual cells are the ones sensitive to light.

The Optic Commissure.—This, called also the chiasma, is formed by the union of the optic tracts in their course forward. It rests on the optic groove of the sphenoid bone. Above, it is attached to the brain, and is separated from the third ventricle by a gray membrane, the lamina cinerea. External to the chiasma is an area pierced by a number of small arteries, the anterior perforated spot. Behind it is the posterior perforated spot and a conic membranous process, the infundibulum, connected with the pituitary body. The chiasma consists of the true and the accessory tracts. The true tracts are of two kinds: the crossing fibres from the nasal sides of the retinæ, which occupy the centre, and the uncrossed fibres from the temporal sides of the retinæ, which occupy lateral positions. The fibres originating in the macula lutea have been so carefully traced of late years as to call for the naming of a third class of fibres, the "macular fascicle." Some of these decussate, while others do not. The accessory fibres are those of Meynert and Van Gudden. They are fibres arising from the internal geniculate body of one side, which, passing forward to the optic chiasma, traverse its posterior border, and then bend backward to end in the internal geniculate body of the other side. They play no part in vision. While the commissure of Van Gudden lies within the posterior angle of the chiasma itself, and is only with difficulty differentiated from the decussated optic fibres, the fibres of Meynert are distinctly separated from the chiasma by a thin layer of gray substance. Their course, too, runs parallel to the posterior angle of the chiasma.

Crossing of the Optic Fibres.—Each optic nerve consists of two bundles of fibres: an outer and an inner. The outer, consisting of fibres coming from the outer half of the retina, do not cross, but pass to the same side of the brain; the inner, derived from the inner half of the retina, cross to the opposite side of the cerebrum and end in the pulvinar, anterior quadrigeminate, and external geniculate bodies. Thus, the right pulvinar, right anterior quadrigeminate, and external geniculate bodies receive fibres from the nasal half of the left eye and the temporal half of the right eye. Thus, the decussation of the chiasma is not complete, but is a semidecussation. Owing to the manner of crossing, the right halves of both retinæ and the left halves of the visual fields belong to the right

optic tract, while the left halves of both retinæ and the right halves of both visual fields belong to the left tract. Objects situated to the left of the observer become known to him by excitation of the right occipital cortex, and *vice versa*. The fact of semidecussation of the optic nerves permits the explanation of half-sight (hemiopia).

Orbital Portion of the Optic Nerve.—The orbital part of the nerve is a firm, white, rounded cord, 3 centimetres in length and 5 millimetres in diameter. It does not pass straight to the eyeball, but forms an S-shaped bend, thus permitting an extensive range of movement of the globe. In passing through the optic canal the nerve lies on the outer side of the ophthalmic artery. In the canal the nerve is liable to injury from fracture of the canal-walls or from growths or suppuration in the sphenoidal sinus.

The *nerve-trunk* consists of over 40,000 medullated fibres supported by a framework of neuroglia. The fibres are disposed in parallel bundles,

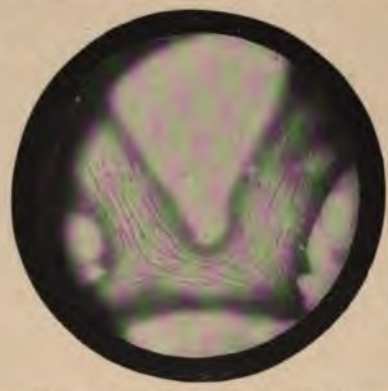


Fig. 46.—Photomicrograph of the optic chiasma, showing decussation of the nerve-fibres. (Author.)

(Photographed by Dr. Carl Fisch.)

which anastomose with each other. Lymph-channels are found between the outer surface of a bundle and the inner surface of the septa. The *nerve-sheaths* in the orbit are three: dural, pial, and arachnoidal. The sheaths are continuous with the cerebral membranes posteriorly and with the sclera in front. The dura forms a thick, loose covering, and is lined with endothelium. Between it and the inner sheath is the *intervaginal space*. The arachnoidal sheath is a delicate layer attached to the dura and pia by trabeculæ of connective tissue. It divides the intervaginal space into an outer, subdural, and an inner, subarachnoidal space, which connect with the intracranial spaces of the same name. The walls of these spaces are covered with endothelium and the spaces are regarded as lymph-channels. The presence of these spaces explains the occurrence of swelling of the head of the optic nerve (papillitis) in certain diseases of the brain. The *blood-vessels* pass from the pia into the nerve; in addition, the anterior part of the nerve transmits the arteria centralis retinæ, which is a branch

of the ophthalmic and enters the nerve from 10 to 12 millimetres behind the eyeball. The central vein empties into the superior ophthalmic vein or into the cavernous sinus. Both artery and vein run in the axis of the nerve to reach the retina. Within the eyeball they are called the central retinal artery and vein.

The optic nerve enters the eyeball through a funnel-shaped opening in the sclera, about 3 millimetres to the nasal side of the optic axis and 1 millimetre below the horizontal meridian. On entering the globe the sheaths and septa are merged into the sclera and the medullary coverings are lost. Hence only the bare axis-cylinders enter the eyeball, and the nerve becomes transparent and of less diameter as it terminates in the optic papilla. At the level of the sclera is a network of connective tissue called the lamina cribrosa. It marks the depth that the observer can see with the ophthal-

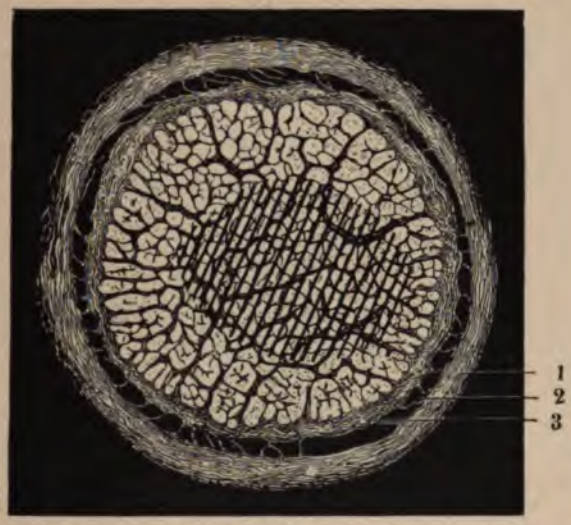


Fig. 47.—Transverse section of orbital part of the optic nerve behind the entrance of the central vessels. (After MERKEL.)

The shaded area marks the position of the papillo-macular fibres. *1*, Dura. *2*, Intervaginal space. *3*, Pia.

moscope. The point of entrance of the optic nerve into the globe is the weakest part of the eyeball. It is this part which first gives way when intra-ocular tension becomes increased. A circle of blood-vessels surrounding the nerve-head makes this one of the most vascular parts of the eye, and communicates with chorioid, sclera, optic sheath, and retina. It is called the circle of Zinn or of Haller.

RADIATIONS OF THE NERVES OF THE EYE.

Optic Nerve.—The peripheral neuron of this nerve is imbedded altogether in the ganglion-cell layer of the retina, while the central neuron originates in the internal layers. The nerve-fibres proceed along the optic

nerve toward the central organ. In the chiasma these fibres partially decussate, so that those of the left halves of the retinæ are distributed to the left tract and those of the right halves to the right optic tract. The optic tracts consequently contain fibres of both optic nerves. It is asserted that long and short tracts exist, of which the long tracts proceed to the lateral geniculate bodies and from there to the posterior limb of the internal capsule. Continuing to the occipital lobes, they form the optic radiation. The short tracts, predominating in numbers, ramify terminally around the ganglion-cells in the corpora quadrigemina anteriora, the corpora geniculata lateralia, and in the pulvinar of the thalamus. A third, or central cortical neuron, from these ganglion-cell fibres passes out to the radiation in the posterior limb of the internal capsule and to the cuneus in the occipital region.

Fig. 48.—Transverse section of the orbital part of the optic nerve in front of the entrance of the central vessels. (After MERKEL.)

The shaded area marks the position of the papillo-macular fibres.

About the meaning of another class of fibres starting from the corpora quadrigemina anteriora, and after decussation going to the retina (centrifugal fibres), nothing certain is known.

Oculomotor Nerve.—The central neuron of this nerve is most likely to be looked for in the gyrus angularis. Running through the base of the internal capsule, it reappears as a part of Spitzer's bundle. It then undergoes decussation in the motor oculi nucleus of the opposite side (beneath the aqueduct of Sylvius). Here its terminal ramification takes place. From the ganglion-cells of this nucleus the roots of the motor oculi are sent out, which, after a partial decussation, run to the eye as the trunk of the motor oculi nerves.

Patheticus Nerve.—The central neuron is identical in its course with that of the preceding nerve. Decussating, its fibres come to their terminal ramification in the trochlear nucleus behind the nucleus of the oculomotorius, below the corpora quadrigemina posteriora. Behind the latter, after decussation, the peripheral neuron passes out as the trochlear nerve.

Abducens Nerve.—The ramification of its central neuron, which pursues a course like that of the foregoing nerves, takes place after decussation in the abducens nucleus under the floor of the fourth ventricle. The abducens nerve (as a peripheral neuron) takes its origin from the ganglion-cells of this nucleus and takes its course through the posterior part of the pons.[1]

THE CRYSTALLINE LENS.

This structure, which is an important part of the accommodative apparatus of the eye, is a transparent biconvex body which rests in the patellar fossa of the vitreous behind, is connected laterally with supporting fibres of the zonula of Zinn, and in front forms a support for the iris. The rounded edge of the lens is separated from the ciliary processes by a space of 0.5 millimetre. The lens presents for examination a central substance, the lens proper, and a covering, the lens-capsule. The lens is unique among optical apparatus in that it has the property of changing its form, thus producing greater or less refraction of light.

Dimensions of the Lens.—The following table, giving the diameters of the lenses of a series of fetal eyes, is by Treacher Collins:—

Age.	Number of Eyes Examined.	Antero-Posterior Diameter.	Transverse Diameter.
4th month	3	2.8 millimetres	3.3 millimetres
5th "	1	3.5 "	4.0 "
6th "	4	3.8 "	4.5 "
7th "	8	4.0 "	5.0 "
9th "	3	4.3 "	5.75 "

One of the measurements of the adult lens is given incorrectly in numerous works on anatomy and ophthalmology, the antero-posterior diameter calculated by Helmholtz (3.7 millimetres) having been often copied. As Priestley Smith has shown, this diameter is 4.5 to 5 millimetres, and in old people is sometimes 6 millimetres or more. Transversely, the lens measures about 9 millimetres. Its weight is 0.20 gramme; its volume is 0.25 cubic centimetre; its specific gravity is 1.121. Its anterior radius of curvature is 10 millimetres for far and 6 millimetres for near

[1] For a full consideration of the neuronic architecture of the visual apparatus the reader can consult the classic work of Dr. St. Bernheimer, of Innsbruck: "Die Wurzelgebiete der Augennerven, ihre Verbindungen und ihr Anschluss an die Gehirnrinde," in the new Graefe-Saemisch "Handbuch der Augenheilkunde," Leipzig, 1900. A chart explanatory of Dr. Bernheimer's researches has been published by Dr. Louis Stricker, of Cincinnati, O., in the *Journal of the American Medical Association*, March 2, 1901.

vision. Its anterior pole is 2.3 millimetres behind the cornea. Chemically the lens consists of 60 per cent. water, 35 per cent. soluble and 2.5 per cent. insoluble albuminoids, 2 per cent. fat with traces of cholesterin, and 0.5 per cent. ash.

Structure of the Lens.—The lens consists of a soft, compressible, non-vascular material which is colorless in youth, but yellowish and opalescent

Fig. 49.—Isolated lens-fibres. (After J. ARNOLD.)

in old age. In the early period of life it possesses a uniform consistency, but in the aged a central hard portion, or nucleus, is distinguishable from the peripheral, or cortical, part, which is soft. Nutrition of the lens is accomplished by the intercellular movement of nutritive fluids. When divested of its capsule, the lens shows on its anterior and posterior surfaces numerous faint, white lines, which radiate from the poles. In the fetal lens there are three of these lines (radii lentis); in the adult they are more numerous.

Fig. 50.—Transverse section of lens-fibres. (After J. ARNOLD.)

They mark the points of junction of the ends of the lens-fibres. The *lens-fibres* are cells which look like bands of ribbon on the flat, and show hexagonal outlines when cut transversely. The superficial fibres are broader and thicker than the central ones. The fibres are 0.005 millimetre broad. They adhere by their edges, which often are serrated in such a way as to leave very minute intercellular spaces for the passage of fluid. The fibres pass in a curved direction in such a way that no fibre reaches from pole to

pole. Those fibres, for example, which begin at the posterior pole end on the anterior surface near the margin. Between the capsule and the lens-substance is found an albuminous material which during life is probably

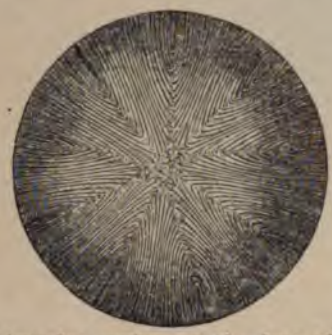


Fig. 51.—Anterior surface of the lens of an adult.
(After J. ARNOLD.)

semifluid, but which after death rapidly liquefies. It is called *liquor Morgagni*. The growth of the lens, after its primary development, comes from the addition of layers of new lens-fibres derived from the cells of the epithelium of the anterior capsule. When incised the lens is seen to be com-

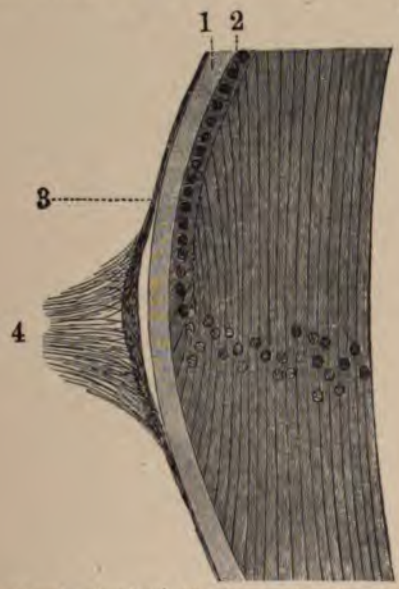


Fig. 52.—Marginal whorl of the lens, showing the transition of the epithelium into lens-fibres. (SCHULTZE.)
1, Capsule. *2*, Epithelium of the capsule. *3*, Layers of the zonula. *4*, Zonula.

posed of lamellæ which can be lifted up and which resemble the layers of an onion. Section of the marginal portion of the lens shows the lens-whorl of O. Becker (Fig. 52), which is due to variations in the axes of developing lens-fibres.

The Lens-capsule is a transparent, elastic membrane which incloses the lens proper and affords attachment to the zonula. The anterior surface of the capsule is about 0.015 millimetre thick, while the posterior is only half this thickness. The inner surface of the front part of the capsule shows a single layer of cubical, polygonal, nucleated cells *(epithelium of the lens-capsule)*. Near the margin of the lens these cells become converted into young lens-fibres.

The Zonula is the suspensory apparatus by which the lens is held in position. It also is an important agent in accommodation, since, when the ciliary muscle contracts, the zonula relaxes and permits the lens to become more convex. To obtain a clear idea of the zonula, the student should harden an eye in formalin (5 per cent.), cut away the posterior half of the globe, and remove the vitreous; then he should remove the cornea and adjacent part of the sclera and cut the iris close to its ciliary border. The zonula is seen, lying in the space between the margin of the

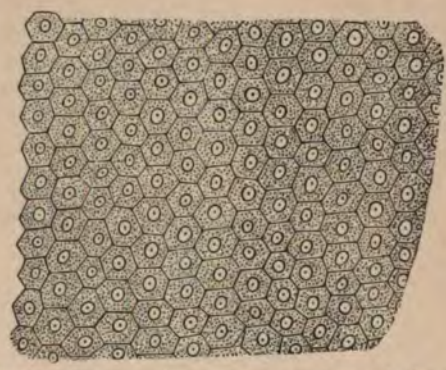


Fig. 53.—Epithelium of the inner surface of the anterior capsule. (SCHULTZE.)

lens and the ciliary processes (Fig. 54), appearing as numerous radiating fibres. The zonula thus formed is a fibrous, reticulated structure, which extends from the ciliary part of the retina to the lens. That part of the zonula which passes from the ciliary body to the lens is known as the *suspensory ligament*. When the ligament is torn away, some of the pigment of the ciliary body adheres to it.

THE VITREOUS BODY.

The greater part of the eyeball, comprising the space between the lens and retina, is filled with a transparent, jelly-like substance called the vitreous humor. It is inclosed in a delicate capsule called the hyaloid membrane. The vitreous body presents anteriorly a central depression, the patellar fossa, in which the lens rests. Posteriorly it is perforated by a lymph-space, the hyaloid canal (canal of Cloquet or of Stilling), which runs from the optic disc to the lens and marks the position of the hyaloid

PLATE II.

Normal Fundus Oculi.

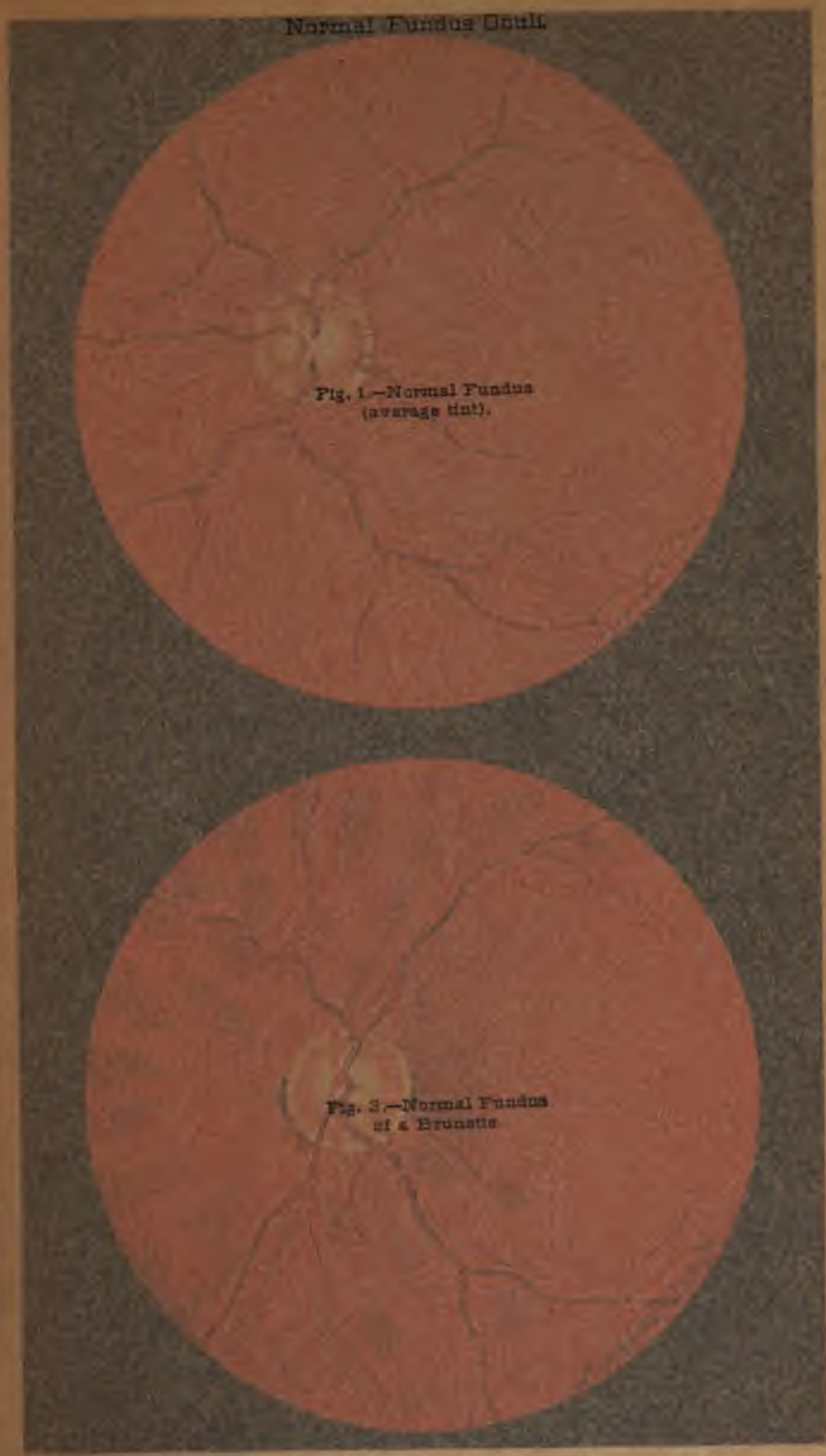


Fig. 1.—Normal Fundus (average tint).

Fig. 2.—Normal Fundus of a Brunette.

Fig. 53.—Epithelium of the inner surface of the anterior capsule. (Stöhr.)

lens and the ciliary processes (Fig. 54), appearing as numerous radiating fibres. The zonula thus formed is a fibrous, reticulated structure, which extends from the ciliary part of the retina to the lens. That part of the zonula which passes from the ciliary body to the lens is known as the *suspensory ligament*. When the ligament is torn away, some of the pigment of the ciliary body

THE VITREOUS BODY.

The greater part of the eyeball, comprising the space between the lens and retina, is filled with a transparent, jelly-like substance called the vitreous humor. It is inclosed in a delicate capsule called the hyaloid membrane. The vitreous body presents anteriorly a central depression, the patellar fossa, in which the lens rests. Posteriorly it is perforated by a lymph-space, the hyaloid canal (canal of Cloquet or of Stilling), which runs from the optic disc to the lens and marks the position of the hyaloid

PLATE 2

artery of intra-uterine life. The vitreous forms a support for the retina, and this function is of greater importance than its action in refraction. Its index of refraction is low (1.336). Chemically the vitreous consists of 98.5 per cent. water, with traces of solids, salts, extractives, proteids, and nucleo-albumin. The fetal vitreous shows a network of interlacing fibrillæ, and is regarded as an embryonal form of connective tissue.

The hyaloid membrane, whose existence has been denied by such eminent observers as Henle, Merkel, and Iwanoff, forms a delicate investment over the whole vitreous body. These structures possess no blood-vessels in adult life, and are nourished by transudation from the vessels of the retina and the ciliary body.

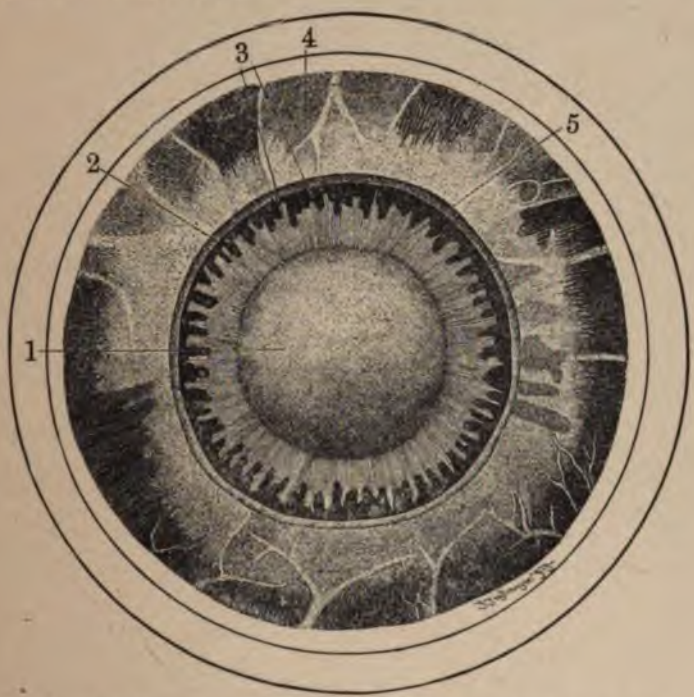


Fig. 54.—Dissection to show the zonula. (After SCHULTZE.)

1, Lens. *2*, Cut surface of the iris. *3*, Ciliary processes. *4*, Chorioid. *5*, Zonula.

In the substance of the vitreous different forms of corpuscles are found, some vacuolated, others not. From them processes project which show bead-like enlargements.

THE OCULAR LYMPH-SPACES.

In the conjunctiva lymphatic vessels are present. Elsewhere in the ocular structures their places are taken by lymph-spaces, which form two systems: an anterior and a posterior.

Lymph from the anterior segment of the globe collects in the anterior and posterior chambers, whence it passes through the ligamentum pecti-

natum into the canal of Schlemm. From this channel it passes into the anterior ciliary veins.

The posterior lymph-spaces are: (1) the hyaloid canal; (2) the perichorioidal space, situated between the chorioid and sclera, and communicating by means of spaces around the venæ vorticosæ with (3) the space of Tenon, which lies between the sclera and Tenon's capsule. From these points lymph collects and passes into (4) the intervaginal space found between the sheaths of the optic nerve and (5) the supravaginal space which surrounds the sheaths of the same nerve. Lymph-spaces sur-

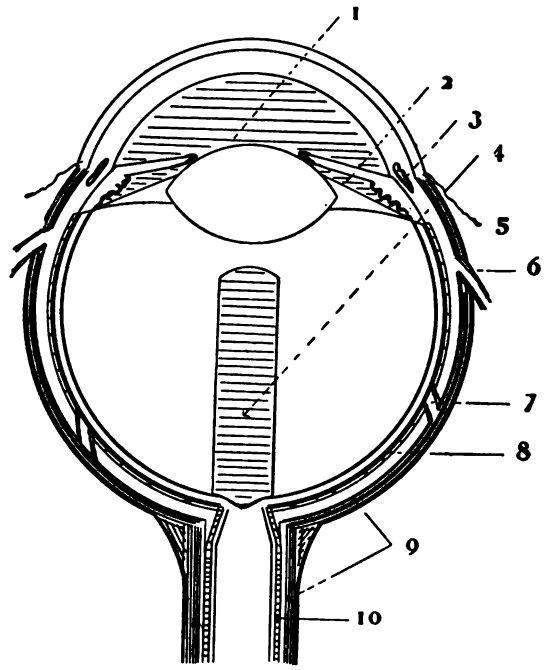


Fig. 55.—Diagram of lymph-spaces of the eyeball. (After FUCHS.)

1, Anterior chamber. *2*, Posterior chamber. *3*, Canal of Schlemm. *4*, Hyaloid canal. *5*, Anterior ciliary vein. *6*, Continuation of Tenon's capsule on the ocular tendons. *7*, Lymph-space around vena vorticosa. *8*, Perichorioidal space. *9*, Supravaginal space. *10*, Intervaginal space.

round the retinal veins and capillaries and probably the arteries. Occlusion of the anterior lymph-spaces is one of the phenomena of glaucoma; nothing is known concerning occlusion of the posterior spaces.

THE NORMAL FUNDUS OCULI.

The optic is the only nerve which can be examined during the life of the patient without dissection. By means of the ophthalmoscope the interior of the eye can be studied. The parts of chief interest in the fundus are the optic disc, the blood-vessels, the macula lutea, and the chorioid.

PLATE III.

Normal Fundus Oculi.

Fig. 1.—Normal Fundus with Unusual Arrangement of Blood-vessels.

Fig. 2.—Fundus of an Albino.

Fig. 10.—Diagram of lymph-spaces of the eyeball. (After Fuchs.)

3, Hyaloid canal. 4, Hyaloid canal. 5, Lymph-space between the sclera and Tenon's capsule. 6, Lymph-space between the sclera and Tenon's capsule. 7, Lymph-space between the sclera and Tenon's capsule. 8, Lymph-space between the sclera and Tenon's capsule. 9, Perivascular space. 10, Intervaginal space.

the retinal veins and capillaries and probably the arteries. Occlusion of the lymph-spaces is one of the phenomena of glaucoma; another is the resulting occlusion of the posterior spaces.

THE NORMAL FUNDUS OCULI.

The optic nerve is the only nerve which can be examined during the life of the patient without dissection. By means of the ophthalmoscope the interior of the eye can be studied. The parts of chief interest in the fundus are the optic disc, the blood-vessels, the macula lutea, and the choroid.

PLATE 3

The Optic Disc.—This is situated about 3 millimetres to the nasal side of the posterior pole of the eye, and is the point of entry of the optic nerve into the retina. It is often called the head of the optic nerve. It measures from 1.4 to 1.7 millimetres in diameter and is generally circular or ellipsoidal in shape. In the astigmatic eye the optic disc often appears oval or ellipsoidal when in reality it is round. Owing to the magnification when the ophthalmoscope is used, the papilla appears to be from 9 to 18 millimetres in diameter. Near its centre is a depression, the physiologic excavation, which marks the divergence of nerve-fibres. The excavation is funnel-shaped, the base being anterior. A trace of the hyaloid artery of fetal life is occasionally seen here as a thread of connective tissue running from the papilla into the vitreous. Surrounding the papilla are two rings: an inner, due to exposure of the sclera, is whitish, and is called the scleral ring; and an outer one, due to the showing of chorioidal pigment, is named the chorioidal ring. At the bottom of the excavation a few dark spots are seen, from the gray stippling of the lamina cribrosa. In color the papilla

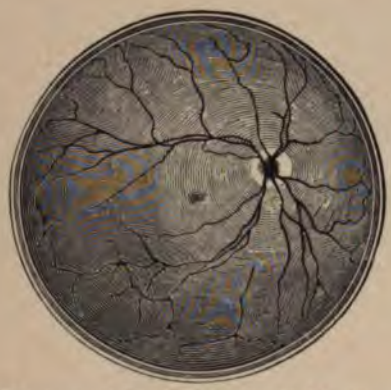


Fig. 56.—The normal fundus of the right eye. (HENLE.)

is grayish pink or reddish, and stands out in marked contrast to the reddish yellow of the remaining parts of the fundus. The color of the papilla varies with the age and complexion of the individual, the color of the surrounding parts of the fundus, and with the illumination used. A bluish discoloration of the disc has been observed as a congenital abnormality. A more common anomaly is the presence of opaque nerve-fibres, which condition is due to the fact that the medullary covering of the axis-cylinders exists in the fibre-layer of the retina. In such a case the fundus shows a patch of a brilliant white color extending out from the disc (Fig. 2, Plate IV). Generally the white area is in contact with the disc. It rarely occurs that the opaque fibres are found at a great distance from the nerve-head or that they occupy a large area of the fundus. The physiologic cup or depression may occupy a large part of the nerve-head, but never extends to the scleral ring. Under normal conditions many variations are seen in the size and depth of the cup and in the arrangement of the blood-vessels.

The Blood-vessels are the central artery and vein. They run in the nerve-fibre layer of the retina, and, although often presenting variations, are of sufficiently regular distribution to justify the naming of the following branches: Superior and inferior nasal, superior and inferior temporal, and macular. The retinal arteries are terminal arteries, each arteriole supplying its own territory without anastomosis. Hence, if a branch is obstructed by an embolus, its territory becomes ischemic and vision is lost. (While this statement is true for almost all cases, in a few instances of embolism of the central artery anastomoses have occurred.) The middle of the fovea centralis has no blood-vessels.

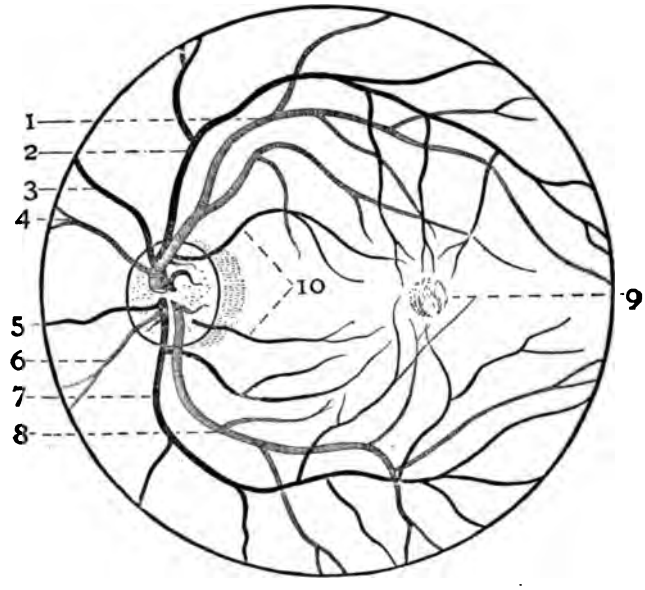


Fig. 57.—Diagram of the retinal vessels of the left eye.

1, Superior temporal artery. *2*, Superior temporal vein. *3*, Superior nasal vein. *4*, Superior nasal artery. *5*, Inferior nasal vein. *6*, Inferior nasal artery. *7*, Inferior temporal vein. *8*, Inferior temporal artery. *9*, Macula lutea. *10*, Macular veins.

While it is often stated that the retinal vessels can be seen on ophthalmoscopic examination, as a fact it is the column of blood, and not the vessel-wall, which is visible. In the larger retinal vessels the blood-column in the arteries is brighter than that in the veins. In the smaller branches this difference is less marked. The brighter color of the arteries is due to the presence of a central streak of light, which is less marked in the veins. The cause of this light-streak is not definitely known. The retinal arteries never pulsate under normal conditions. (To this statement, which is true for the vast majority of individuals, exceptions must be made, since Jaeger, von Graefe, Donders, and other competent observers have seen spontaneous arterial pulsation in normal eyes.) The reason for non-pulsation in the retinal arteries is this: the normal intra-ocular tension

PLATE IV.

Normal Fundus Oculi.

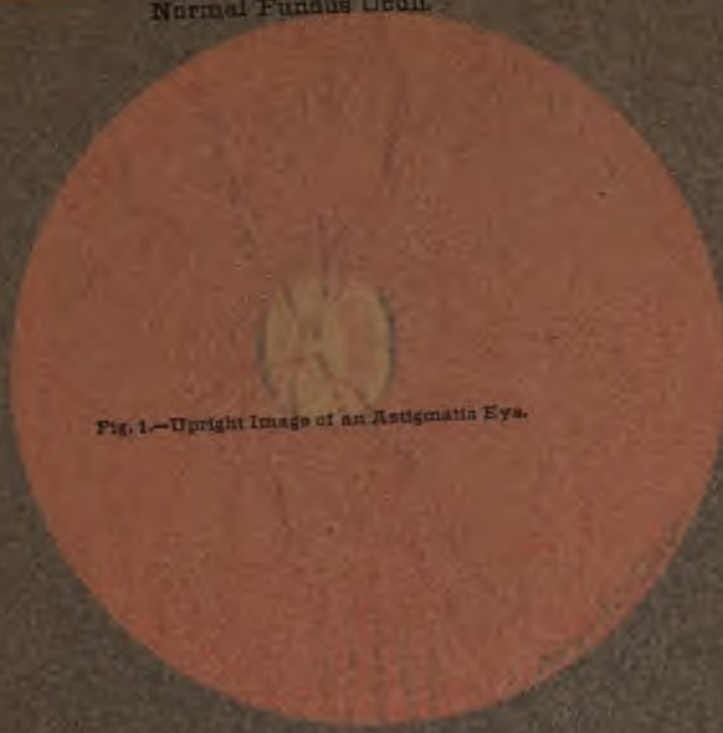


Fig. 1.—Upright Image of an Astigmatic Eye.

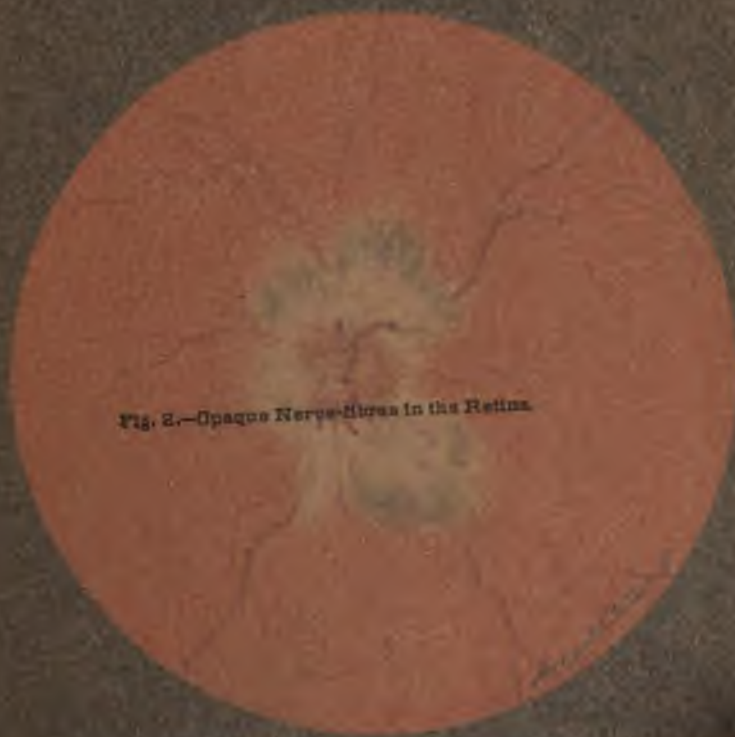
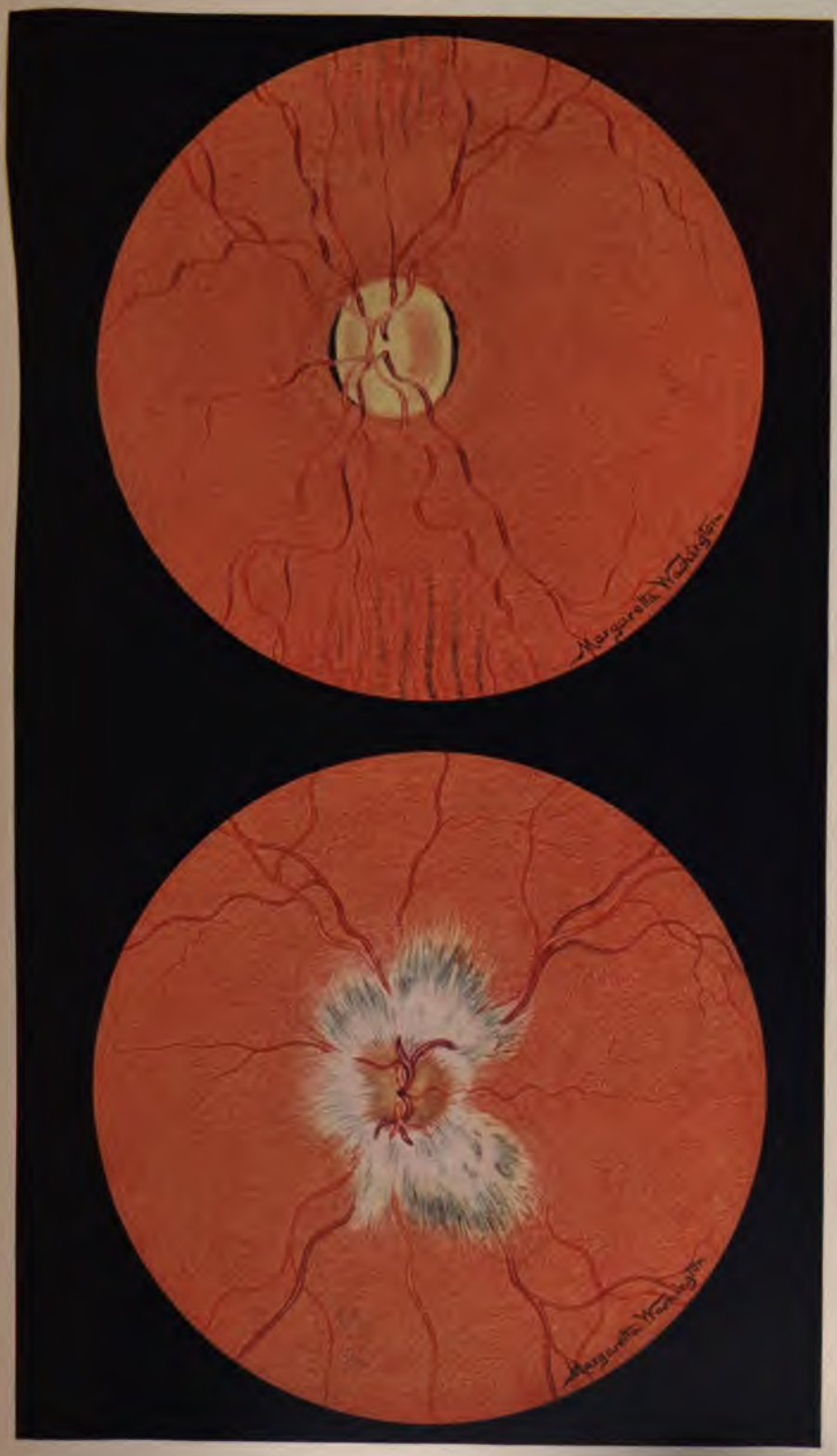


Fig. 2.—Opaque Nerve-fibres in the Retina.

9

PLATE 4



is sufficient to overcome the diastole of the heart. Arterial pulsation may be produced easily in the normal eye by pressure on the globe. Whenever a disproportion exists between intra-ocular and intra-arterial pressure, arterial pulsation occurs. Venous pulsation occurs spontaneously in from 60 to 75 per cent. of normal eyes.

Besides the blood-vessels enumerated above, it is necessary to mention the cilioretinal vessels. These are commonly small, solitary vessels which arise from the circle of Haller, and emerge at the temporal border of the disc. Such a vessel may come from the central vessel in the substance of the nerve, and may be of larger size. Generally it supplies blood to a small area between the disc and macula. Cilioretinal vessels are present in from 10 to 16 per cent. of normal eyes. Their presence has been known to permit a portion of the retina to retain its functions in cases of embolism of the central retinal artery. Most cilioretinal vessels are arteries.

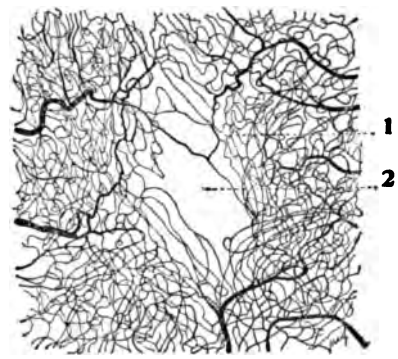


Fig. 58.—Injected blood-vessels of the macular region.
(BÖHM and DAVIDOFF.)
1, Capillary loops of the macular region. *2*, Fovea centralis—no vessels.

Having described the usual arrangement of the blood-vessels, it is necessary to mention some of the unusual appearances found in normal eyes. Twisting of a vein and artery often occurs; but it rarely happens that an artery crosses an artery, or a vein crosses a vein. Anastomoses are very rare anomalies, and occur on the optic disc. Instances of bifurcating arteries and veins are shown in several ophthalmoscopic atlases. Although the retinal vessels do not pursue a straight course, their tortuosity is subject to much variation. A rare anomaly is the presence of a projecting loop. In Lawford's case a vein formed a loop, each end of which disappeared in the disc. An unusual arrangement of vessels observed by the author is shown in Fig. 1, Plate III. Here incomplete twisting of the superior temporal artery and vein is seen; cilioretinal arteries are present at the nasal side of the disc; and the arterial supply of the lower half of the retina comes from a large trunk, which divides into four branches at the lower margin of the disc. The case was that of a boy, aged 12 years, whose vision was normal after the correction of a small amount of hyper-

metropia. The presence of a persistent hyaloid artery, as a small loop projecting into the vitreous humor, is occasionally observed. Its persistence as a blood-carrying vessel is a rare abnormality.

Perivascular Lymph-channels.—His (1865) was the first anatomist to demonstrate the existence of perivascular lymph-spaces in the retina. Schwalbe, Norris, and others, by injecting fluid beneath the pial sheath of the optic nerve, have filled the spaces around the retinal veins and capillaries. Analogous spaces surround the retinal arteries.

The Macula Lutea.—Situated about 3 millimetres to the outer side of the optic-nerve head, and slightly below the horizontal meridian, is a spot darker than the surrounding retina and apparently devoid of blood-vessels. This is the macula lutea (yellow spot). It is the area of greatest visual acuity. The centre of the macula presents the foveal reflex, while the periphery shows a whitish, glistening ring, or halo, known as the macular reflex. It is strange that no two ophthalmic writers agree as to the color and appearance of a part of the retina so accessible to examination as the macula, and that the errors of forty years ago should appear in modern text-books. Many writers have portrayed the macula as oval, with its long diameter placed transversely. Schmidt-Rimpler described it as anatomically circular, but ophthalmoscopically oval. Panas and Mauthner saw it as a brilliantly silvered ring. Power spoke of it as "a soft, whitish line"; and Landolt described it as "a bright, oval line, sometimes glistening, with a red floor and intensely red, almost black, centre, the dark point in the centre being hardly ever absent." These differences in appearance are doubtless due to several causes: the difference in methods of examination; in the age, complexion, and refraction of different individuals; and variations in the distribution of pigment. Johnson states that, when observed in a certain way, the macular ring in its whole circumference can be seen in every person under thirty-five years of age, and frequently in older subjects. If the illumination is lowered, reflection from the fundus decreases more rapidly than from the macula, until a moment arrives when the ring appears. He asserts that the macular ring is invariably circular, and probably corresponds to the extreme limit of the macular region. When observed as an oval, the appearance is due to distortion produced by the lens and mirror. When examined carefully by the direct method of ophthalmoscopy the macula is always round. In elderly persons it can be recognized, although with more difficulty than in the young, by its darker color and by the absence of vessels.

There are several forms of macular rings. Johnson states that the most common is a bright, scintillating reflex resembling shot-silk, very marked in dark eyes, scarcely visible in fair ones, and best seen with feeble illumination. This ring is supposed to be due partly to reflection from Müller's fibres, where they expand into the internal limiting membrane, partly to the fibrous sheaths of the vessels which lift up the retina overlying them. A second form of ring is a radiating circle of grayish-white

PLATE V.

Normal Fundus Oculi: Macular and Foveal Reflexes. (JOHNSON.)

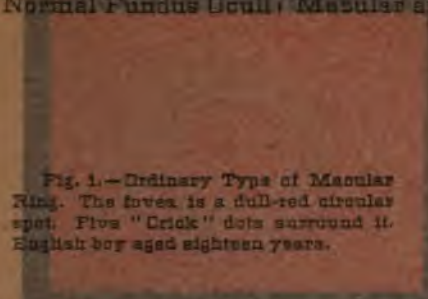


Fig. 1.—Ordinary Type of Macular Ring. The fovea is a dull-red circular spot. Five "Crick" dots surround it. English boy aged eighteen years.

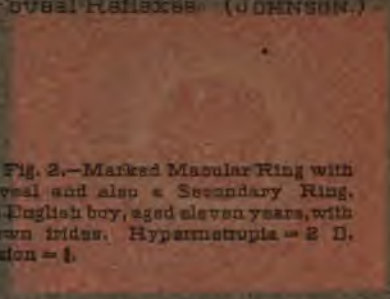


Fig. 2.—Marked Macular Ring with Foveal and also a Secondary Ring. An English boy, aged eleven years, with brown irides. Hypermetropia = 2 D. Vision = 1.

Fig. 3.—Macular Region of a Native African Boy, Aged Sixteen Years, from the Lower Congo Region.

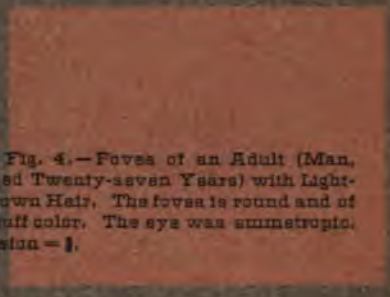


Fig. 4.—Fovea of an Adult (Man, Aged Twenty-seven Years) with Light-Brown Hair. The fovea is round and of a buff color. The eye was emmetropic. Vision = 1.

Fig. 5.—A Long "Comet-flare" from a Boy, Aged Eight Years, with Light Flaxen Hair. Vision = 1.

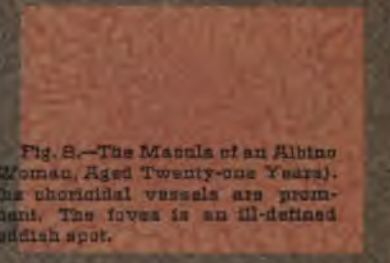


Fig. 6.—The Macula of an Albino (Woman, Aged Twenty-one Years). The choricidal vessels are prominent. The fovea is an ill-defined reddish spot.

Fig. 7.—Macular and Foveal Rings. Spanish girl with black hair and dark-brown irides. Hypermetropia = 1 D. Vision = 1.

Fig. 8.—Macular Region of a Woman, Aged Thirty-six Years, with Brown Hair, Gray Eyes, and Fair Complexion. The whole fundus is stippled with black specks like grains of pepper. Vision = 1.

lustre, the radii being directed toward the fovea and resembling nerve-fibres. The appearance is supposed to be due to a partial translucency of the nerve-fibres. The third form of ring can be seen with the brightest illumination as a whitish or golden ring of metallic lustre, oval in shape by indirect ophthalmoscopy, but circular when seen by the direct method. It is narrower than the other two rings.

The foveal reflex is found in the centre of the macula as a very small ring, or as a circular or horseshoe-shaped spot of light, or as a "comet-flare." It is due to reflection of the edge of the fovea.

The Chorioid.—While each ocular tunic contributes something to the ophthalmoscopic picture, the chief part must be credited to the chorioid. Light reflected from the mirror of the ophthalmoscope passes through the transparent part of the retina to the pigment epithelium, and is partly absorbed, partly reflected. Although the pigment layer belongs embryologically to the retina, it generally adheres to the retinal surface of the chorioid, and is accredited ophthalmoscopically to the latter tunic. The brightness of the fundus picture depends on the amount of pigment. The greater the pigment, the greater the absorption of light and the darker the fundus picture. In the negro and the native of India the fundus is of a brownish, brown-red, or slate color, while in the Anglo-Saxon, and particularly in blondes, it is of a bright-red color. If the pigment layer is very thin, the chorioidal vessels are correspondingly exposed and are seen as a network of large, flat vessels, without a light-streak, between which are spaces of light or darker color. They are seen best in albinos. It is generally impossible to differentiate between the chorioidal arteries and veins, although at the equatorial region the latter converge to form the venæ vorticosæ. In brunettes the vessels appear as "light streams separated by dark islands," because the spaces are more deeply colored than the vessels.

The Sclerotic, which may be spoken of as the panel on which the fundus picture is painted, is commonly invisible, being covered by the nearly opaque chorioid. Yet it is probable that in all eyes some light passes through the chorioid, and thus the sclera has some influence on the ophthalmoscopic picture, serving to make it lighter. In albinos the sclera appears as a white surface between the chorioidal vessels. It is best seen where the chorioid is absent, as in coloboma, or pathologically as a result of destruction of the retina and chorioid.

CHAPTER III.

PHYSIOLOGY OF VISION.

THE sense of sight is the most highly developed and differentiated of the special senses. The eye is for the purpose of receiving rays of light which are focused on the retina, where they are changed into a stimulus of nerve-fibres. These stimuli are transmitted to the brain via the optic nerve. It is necessary to study: (1) the mechanism of the formation of retinal images, and (2) the events following the formation of images.

MECHANISM OF THE FORMATION OF IMAGES.

Refraction.—In order to understand how an image is formed on the retina, it is necessary to mention a few optical principles with reference

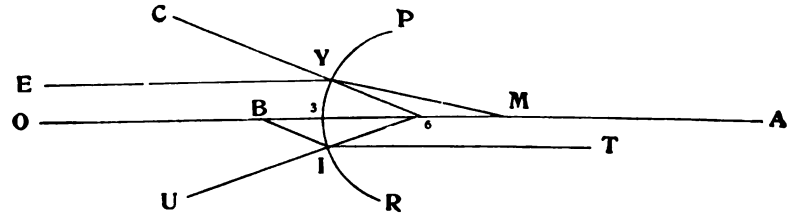


Fig. 59.—Diagram of a simple optical system.

particularly to the laws of refraction of light by transparent bodies bounded by curved surfaces. The simplest form of dioptric apparatus consists of two media of different refractive indices separated by a spherical surface. The optical properties of such an apparatus depend on (1) the curvature of the surface, and (2) the refractive powers of the media. Such a simple optical system is shown in Fig. 59.

The line *P-R* represents a curved surface separating media of different refractive power, the less being on the left. The line *O-A*, falling perpendicular to the surface at *3*, passes through the centre *(6)* of the sphere with whose surface we are dealing. This line, *O-A*, is the *optic axis*. All lines which cut the surface normally, such as *O-A*, *C-Y*, and *U-I*, undergo no refraction. They continue in straight lines and cross at *6*, which is the *nodal point*. All other rays passing from a medium of less to one of greater density are refracted. All lines lying in the first medium and parallel to the optic axis will be bent so as to meet at *M*, which is the *principal posterior* (or second) *focus*. On the optic axis, in the first medium, is an important point, *B*. Rays of light passing from *B*, such as *B-I-T*, are so refracted that they become parallel to the optic axis. *B*

is called the *principal anterior* (or first) *focus.* The point where the optic axis cuts the surface is called the *principal point.* The above-mentioned points—posterior and anterior foci, the nodal point, and the principal point—are called the *cardinal points* of an optical system.

It is necessary to know the effect of a biconvex lens upon light, because the dioptric media of the human eye equal such a lens. The action of a

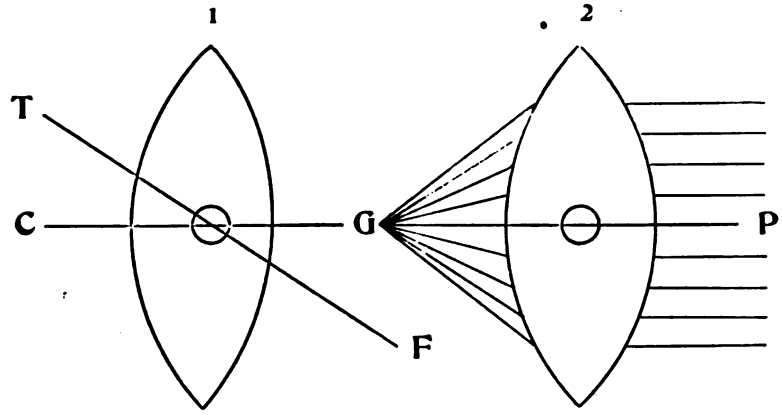


Fig. 60.—Action of a biconvex lens upon rays of light.

biconvex lens can be appreciated by reference to Fig. 60. In the illustration at the left (1), *C* and *G* mark the centres of curvature. The line connecting them is the chief axis, and the centre of this line is the optical centre of the lens. Rays passing through the optical centre of a lens are unbent. The illustration at the right (2) shows that rays falling on a

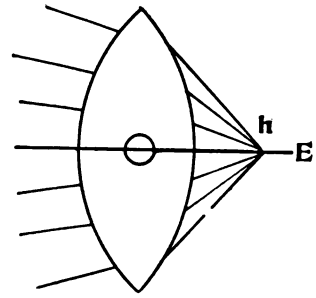


Fig. 61.—Action of a convex lens on rays of light proceeding from a point within the focus.

lens parallel with its principal axis, *G-P,* are collected on the opposite side at a point called the principal focus. Conversely, rays which diverge from the focus after passing through the lens emerge as parallel lines. Fig. 61 shows that rays which pass from a point within the focus *(h)* after passing through the lens leave it less divergent, but do not come to a focus again.

Convex lenses form real and inverted images of such objects as are placed beyond the focal point of the lens. The action of a biconvex lens is explained in Fig. 62. The arrow *M-P*, placed upright before a biconvex lens, gives an inverted image *(p-m)* on the opposite side of the lens.

To form an image on the retina it is necessary that rays of light shall be bent. The amount of bending or refraction of a ray of light depends on: (1) the radius of curvature of the refracting surface; and (2) the difference in the refractive indices of the medium from which the ray comes and that into which it passes. The smaller the radius of curvature and the greater the difference of refractive index, the more the ray will be bent. The refractive media of the eye are the cornea, aqueous humor, lens, and vitreous humor. The following table gives the radii of curvature of the refracting surfaces, the refractive indices of the dioptric media, and other data needed in the study of refraction in the human eye:—

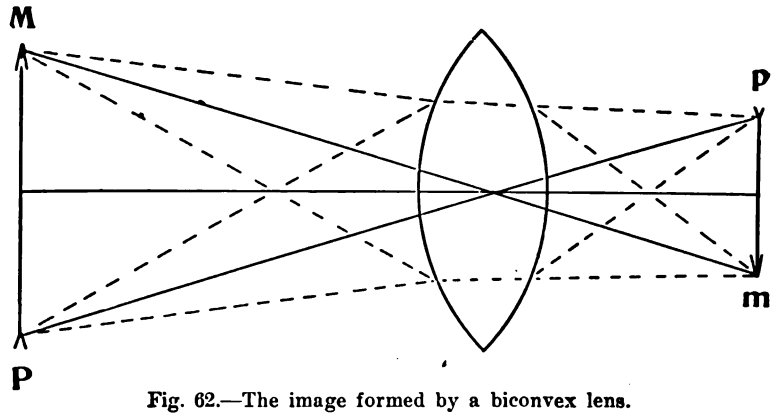


Fig. 62.—The image formed by a biconvex lens.

		In Accommodation for Far Vision.	In Accommodation for Near Vision.
Radius of curvature of	Cornea	7.8 mm.	7.8 mm.
	Anterior surface of the lens	10.0 "	6.0 "
	Posterior surface of the lens	6.0 "	5.5 "
Distance between	Anterior surface of the cornea and the anterior surface of the lens	3.6 mm.	3.2 mm.
	Anterior surface of the cornea and the posterior surface of the lens	7.2 "	7.2 "
	Anterior and posterior surface of the lens	3.6 "	4.0 "
	Posterior surface of the lens and retina	14.6 "	14.6 "
Antero-posterior diameter of the eye along the axis		21.8 mm.	21.8 mm.

Refractive Indices.

Air	1.000	Cornea	1,337
Water	1.335	Vitreous humor	1,3365
Aqueous humor	1.3365	Lens (average)	1,437

The Reduced Eye.—In order to simplify the problem connected with the formation of retinal images, physiologists have constructed schematic and reduced eyes. The measurements given below are for the reduced eye of Listing:—

Radius of curvature of the single refracting surface	5.1	mm.
Index of refraction of the single refracting surface	1,35	
Antero-posterior diameter of the reduced eye	20.0	mm.
Distance of the single refracting surface behind the anterior surface of the cornea	1.8	"
Distance of the nodal point from the anterior surface	5.0	"
Distance of the nodal point from the principal focus (retina)	15.0	"

The Visual Angle.—If the position of the centre of curvature of the single refracting surface of the reduced eye—*i.e.*, the nodal point—is known, the position of the retinal image of an object can be found by drawing straight lines from the circumference of the object through the

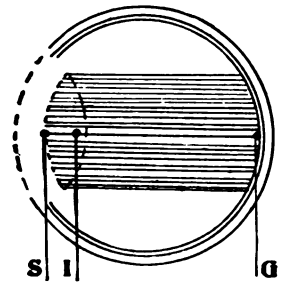


Fig. 63.—Diagram of the reduced eye.

S, The single spheric refracting surface 1.8 mm. behind the anterior surface of the cornea. *I*, The nodal point, 5 mm. behind *S*. *G*, The principal focus (on the retina), 20 mm. behind *S*. The cornea and lens are represented by dotted lines in the positions they should occupy.

nodal point to the retina. These lines cut the refracting surface at right angles and pass through the single refracting surface without bending. The retinal image is found to be inverted. The size of the image is in proportion to the angle made by the lines drawn from the periphery of the object to the nodal point. This angle is called the *visual angle* (Fig. 64). Objects seen under the same visual angle have retinal images of the same size. Acuteness of vision is measured by the smallest angle within which an object is visible. The normal eye recognizes letters five times as long as they are broad when they are at such distance that they strike the eye at an angle of five minutes.

The Retinal Image can be demonstrated in the enucleated eye of an albino rabbit and in the human eye. The image differs from the object in size and in the relative arrangement of its parts. Its size is generally smaller than that of the object, regardless of the distance; and its position is reversed, the lower part of the image representing the upper part of the object, and the left side of the image corresponding to the right side of

the object. In visual judgment, however, as will be mentioned later, the retinal image is reinverted.

The size of the retinal image can be calculated, if the size of the object and its distance from the nodal point are known. The formula for determining the size is this:—

$$\text{Size of retinal image} = \frac{\text{size of object} \times \text{distance of the image from the nodal point}}{\text{distance of the object from the nodal point}}$$

Accommodation.—By this term is meant the power possessed by the eye of seeing objects distinctly at different distances. The agents concerned in accommodation are the lens and ciliary muscle. The eye has often been compared to the camera of the photographer: the dioptric media equal the lens, the pigment of the retina and chorioid makes the chamber dark, the

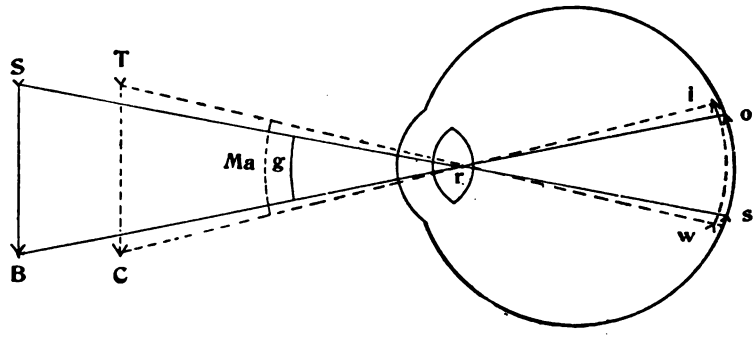


Fig. 64.—Diagram to show that the visual angle and size of the retinal image vary with the distance of the object from the eye.

The image of *S-B* is seen at *o-s*, under the angle *g*; and the image of *T-C* is seen at *i-w*, under the angle *Ma*.

retina is the sensitive plate, the iris regulates the amount of light, and the focus is obtained by the action of the ciliary muscle on the lens. In the camera the focus is obtained by moving the sensitive plate nearer to or farther from the lens. In the human eye the retina occupies a fixed position, while the focusing is accomplished by alterations in the curvature of the lens. As regards the exact mechanism of accommodation authorities differ, there being two principal theories. Helmholtz held that, when the ciliary muscle contracts, the chorioid and ciliary processes are brought forward, the suspensory ligament of the lens is relaxed, and the lens—by virtue of its elasticity—becomes more convex. Tscherning has advanced the view that accommodation does not depend upon slackening of the zonula of Zinn, but on its tension, which is brought about by the ciliary muscle, by which the periphery of the lens becomes flattened and the curve of its surface assumes an hyperboloid form. Tscherning states that he has actually produced this change by making traction on the zonula in the

eyes of animals from which the cornea had been removed. Schoen has also attacked the Helmholtz theory of accommodation. Which of these hypotheses is correct must be decided by future investigations.

That a change takes place in the pupillary part of the lens during accommodation is demonstrated by the alterations occurring in the reflexes called the Purkinje-Sanson images. These are catoptric images (*i.e.*, reflected) from the cornea and anterior and posterior surfaces of the lens. They can be understood by a study of Fig. 65. Let light fall on the eye through a triangular opening. Three images will be seen: the brightest, an erect virtual image, is from the anterior (convex) surface of the cornea; another erect virtual image, larger, but less bright, is from the anterior (convex) surface of the lens; and a small inverted real image is from the (concave) posterior boundary of the lens. The second image is intermediate in position between the other two. If the eye is accommodated for a near point, the corneal image is unchanged. The middle image

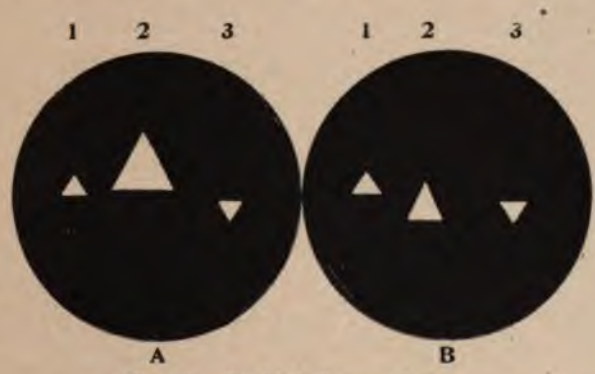


Fig. 65.—Purkinje-Sanson images.

A, In the absence of accommodation. *B*, In accommodation. *1*, Reflection from the cornea. *2*, From the anterior surface of the lens. *3*, From the posterior surface of the lens.

becomes smaller and approaches the corneal image, showing the curvature of the anterior surface of the lens to be increased. A slight change occurs in the third image. By means of special apparatus Helmholtz was able to determine that in maximum accommodation the radius of curvature of the anterior surface of the lens is 6 millimetres as compared with 10 millimetres when the eye is at rest. Another change which occurs in accommodation is a slight sinking of the lens, which is due to its weight (Heine).

The nervous mechanism of accommodation is under the control of the will. The ciliary muscle is governed by fibres which can be traced through the short ciliary nerves and ciliary ganglion, along the third nerve, to a centre located (in dogs) at the front end of the aqueduct of Sylvius. Stimulation of this centre, or of the third nerve, or of the short ciliary nerves leads to contraction of the ciliary muscle and accommodation for near objects.

In recording accommodation certain terms are used which must now

be explained. The most distant point of distinct vision is called *the far point (punctum remotum)* $= R$. The nearest point at which small print can be read is named *the near point (punctum proximum)* $= P$. The amount of accommodative effort of which the eye is capable is called *the amplitude of accommodation* $= A$. In the normal eye the amplitude of accommodation is expressed by the formula: $A = P - R$, in which P and R are expressed in dioptres (a dioptre is a glass whose focal length is one metre). If the distance of the near point is known in centimetres, the equivalent in dioptres is found by dividing 100 by the near point in centimetres. Thus, if the near point be 20 centimetres, the amplitude of accommodation $= 5$ D. $(A = P - R;$ or $A = \frac{100 \text{ cms.}}{20 \text{ cms.}} - R,$ which in this example is 0; hence A $= 5$ D.) The near point *(P)* can be found

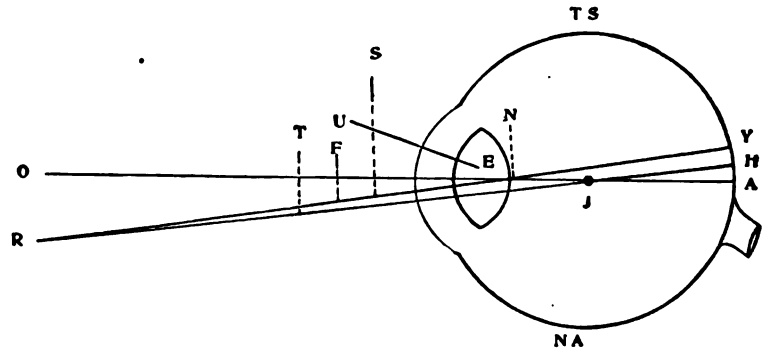


Fig. 66.—Diagram showing the corneal axis, *U-E;* the optic axis, *O-A;* the visual line, *R-Y;* the line of fixation, *R-J;* and the three angles.

The angle between *U-E* and the visual line, *R-Y*, is the angle alpha, averaging five degrees. The angle between the optic axis, *O-A*, and the line of regard, *R-J*, is the angle gamma. The angle between the optic axis, *O-A*, and the line of vision, *R-Y*, is the angle beta. *TS* = temporal side; *NA* = nasal side.

by ascertaining the nearest distance at which the patient can read fine print; the distance is then measured by a steel tape marked both in dioptres and in centimetres.

Optical Defects.—The eye is not a perfect instrument, since it is not exactly centred, and possesses chromatic and spheric aberration in a small degree. In a perfect optical instrument the lenses are exactly centred, and the visual line corresponds to the axis of the lens-system. *The optic axis* of the human eye passes through the centre of the cornea, but not through the centre of the lens, and does not touch the fovea centralis: the region of distinct vision. *The line of vision* is the line connecting the object viewed, the nodal point, and the fovea. *The line of fixation* connects the object with the *centre of rotation,* which is placed 6 millimetres behind the nodal point. The angle included between the major axis of

the corneal ellipse and the visual line is called the *angle alpha,* and measures about five degrees. The angle included between the optic axis and the line of fixation is named the *angle gamma.* The angle between the optic axis and the visual line is named the *angle beta.* These are shown in Fig. 66, and will be referred to in the chapter on refraction.

In the normal (emmetropic) eye the principal posterior focus lies on the retina. The myopic eye has its principal posterior focus in front of, and the hypermetropic eye behind, the retina. These must be consid-

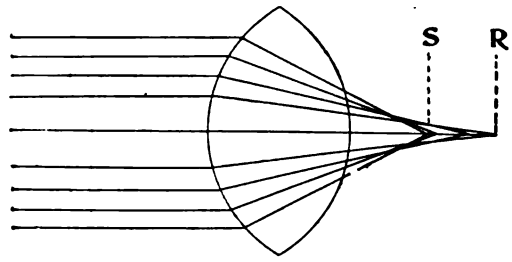


Fig. 67.—Diagram to show spheric aberration.

The distance from *S* to *R* is the focal line of Sturm.

ered as abnormal eyes, although, since at birth and in infancy all eyes are hypermetropic, some writers contend for the adoption of the hypermetropic eye as normal. In the middle and later periods of life presbyopia (old sight) appears, owing to a diminution in the elasticity of the lens or a weakness of the ciliary muscle. All these conditions can properly be considered pathologic, and will be discussed in the chapter on refraction. The normal eye, however, is subject to certain defects: spheric and chromatic aberration and astigmatism.

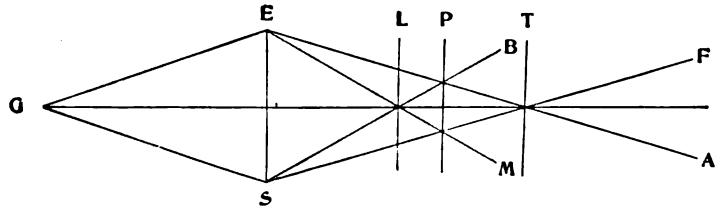


Fig. 68.—Diagram to show chromatic aberration.

SPHERIC ABERRATION.—Rays of light which traverse the peripheral parts of the lens are brought to a focus sooner than those which pass nearer the centre. This inequality is known as spheric aberration. The interval between the foci is known as the focal line of Sturm (Fig. 67). The iris, acting as a diaphragm, shuts off the peripheral rays and corrects this defect.

CHROMATIC ABERRATION.—Different rays of the spectrum are bent to different degrees. Violet rays, being more refrangible than red, will

have their focus nearer to the lens. This inequality is known as chromatic aberration. In the manufacture of optical instruments the defect is overcome by combining a convex with a plano-concave lens, each made of a glass with different refractive index. In the human eye practically the same arrangement of lenses exists, and, owing to ability rapidly to change accommodation, chromatic aberration attracts little notice. That chromatic aberration exists in the eye can be demonstrated by the study of Fig. 68. The chromatic test for errors of refraction is based on chromatic aberration.

In Fig. 68 let it be supposed that *G* is a luminous point, *E-S* the dioptric surface, and *P* to be the plane of the mean focus of *G*. Violet rays, *E-M* and *S-B*, will be brought to a focus in the plane *L;* the red rays, *E-A* and *S-F*, in the plane *T*. If the rays fall on the retina between *L* and *P*, the diverging, or blue, rays will form a centre around which will be the converging red rays, *E-A* and *S-F*. If the rays fall on the retina between *P* and *T*, the converging red rays will form a centre bordered by the diverging blue rays, *E-M* and *S-B*, around them, provided those lines were carried to the plane, *T*.

Astigmatism.—The dioptric surfaces of the eye are not parts of perfect spheres. Slight variations do not cause inconvenience, but marked differences in the curvature of the refracting surface in different meridians will reduce vision. Most eyes possess a small amount of regular corneal astigmatism (regular because the meridians of greatest and least refraction are at right angles), the cornea being of greater convexity in the vertical meridian. Occasionally regular astigmatism is due to the unequal curvature of the lens. Irregular astigmatism, the principal meridians being placed otherwise than at right angles, exists in the normal lens to a slight extent. It is this which causes a star to look, not round, but pointed.

Lack of Transparency.—The dioptric media of the eye are not perfectly transparent, as is shown by the presence of small, bead-like bodies *(muscæ volitantes)* which are seen when one looks through a microscope. They are due to floating particles in the vitreous humor.

Functions of the Iris.—The iris is a diaphragm cutting off light which otherwise would pass through the periphery of the lens and thus cause spheric aberration. It also forms a support for the ciliary muscle in accommodation.

With the eye at rest the pupil enlarges; in accommodation it becomes smaller. The movements of the iris are under the control of a delicate nervous arrangement as follows: The ciliary nerves (branches of the lenticular ganglion) pierce the sclera near the entrance of the optic nerve, pass forward between the lamina fusca and the chorioid, and supply the ciliary muscle, iris, and cornea. They distribute sensory filaments from the fifth nerve to the eyeball; motor fibres to the ciliary muscle and to the sphincter of the iris from the third nerve; and sympathetic fibres from the cavernous plexus, which on irritation cause the pupil to dilate.

The pupil is constantly changing in size, so that no physiologic stand-

ard of measurement can be adopted, although it is generally stated that the average diameter is 4.4 millimetres. It is large in children and small in the aged.

CONTRACTION OF THE PUPIL occurs normally (1) in response to stimulation of the retina by light, and (2) during accommodation. The abnormal conditions causing contraction are numerous, and embrace the poisonous effects of certain drugs and gases and the manifestations of certain nervous diseases, all of which will be considered in the clinical part of this treatise.

DILATION OF THE PUPIL is brought about normally (1) when stimulation of the retina is diminished or absent, as in passing from a brightly lighted to a dark room; and (2) when the eye is directed toward distant objects. Abnormally, contraction occurs in poisoning by some drugs, in certain nervous diseases, and in certain pathologic conditions of the eyeball.

Constriction of the pupil is brought about by the action of the circular muscular fibres found in the pupillary margin of the iris. Dilation is an action whose exact mechanism is still a subject of debate. Some physiologists hold that it is simply a negative act: that, when the sphincter pupillæ relaxes, the elastic radiating fibres of the iris cause the enlargement; while others claim the existence of special dilating fibres.

Contraction of the pupil in response to the stimulus of light falling on the retina is a reflex act, the optic being the afferent nerve, the motor oculi the efferent path, and the centre being situated in the floor of the aqueduct of Sylvius.

Aside from this optic-oculomotor reflex mechanism there are *other agencies which influence the pupil.* Thus, section of the cervical portion of the great sympathetic nerve is followed by contraction. If the upper end of the sympathetic is stimulated, the pupil becomes larger. This influence of the sympathetic upon the iris is the antipode to what occurs to the blood-vessels. When the sympathetic nerve has been sectioned, the vessels of the head and neck become enlarged, nasal and lacrimal secretion are increased, the temperature of the corresponding side of the head is raised, and the pupil contracts. When the sympathetic has been stimulated the vessels contract, but the iris dilates. Excision of the superior cervical ganglion of the sympathetic shows that in the human subject the removal of this part of the sympathetic nerve is followed by dilation of the vessels and contraction of the pupil.

In the lower animals a set of pupil-dilating nerve-fibres can be traced from a centre located in the floor of the front part of the Sylvian aqueduct. Thence it passes through the cervical part of the spinal cord, the anterior roots of the upper dorsal nerves, the upper thoracic ganglion, and the cervical portion of the sympathetic nerve. It can be further traced through a branch of the superior cervical ganglion, which passes upward with the internal carotid artery, and thence over the Gasserian ganglion to the ophthalmic division of the fifth nerve. From this point the nerve-fibres

pass through the nasal branch and the long ciliary nerves to supply the eye. These fibres are constantly active. If sectioned in any part of their course, contraction of the pupil follows; stimulation of the distal end of the divided nerve causes dilation of the pupil. The short ciliary nerves must be regarded as carrying pupil-constricting impulses, while the pupil-dilating impulses traverse the long ciliary nerves.

The optic-oculomotor mechanism adapts the pupil to the amount of light, and the sympathetic mechanism transmits emotional and sensory influences. Dilation of the pupil occurring during fear or during pain is due to the sympathetic.

Certain drugs act on the iris independently of nervous connection. An eye in which the optic, oculomotor, and sympathetic nerves have been cut will still dilate to atropin or contract to eserin. Such drugs act in a local manner on the sphincter of the pupil.

The fifth is the sensory nerve of the iris. It also carries the dilating fibres of the sympathetic.

Associated Action between the accommodation and convergence is interesting. Whenever we look at a near object the eyeballs converge and the pupils become small. When the eye is turned to a distant object the pupil dilates. These are examples of "associated movements."

Monocular Diplopia.—Scheiner's experiment, which was used by its author to prove accommodation, shows that double vision can be had with one eye alone. The experiment is as follows: Take a piece of cardboard, and with a pin make several holes so close to one another that two or more will occupy a space of less diameter than the pupil. Now place the card in front of one eye, closing the other, and look at a pin placed at the reading distance. It will be seen properly, only somewhat dimmed; if brought nearer or (in myopia) placed farther from the eye, it will be seen double. Under some pathologic conditions monocular diplopia occurs.

OCULAR MOVEMENTS AND BINOCULAR VISION.

Movements of the Eye.—The movements of the eye are limited practically to its rotations around a centre which Donders and Dojer found to be 10 millimetres in front of the posterior surface of the sclera, or 14 millimetres behind the summit of the cornea. This is the *centre of rotation.* The six muscles attached to each eyeball cause the globe to rotate around three axes, which pass through the centre of rotation. The axis of the external and internal recti is vertical, while the axes of the other pairs of muscles (superior and inferior recti, superior and inferior obliques) are in the horizontal plane.

In order to avoid confusion it is perhaps well to define the three *planes of separation* of the eyeball: (1) the horizontal plane of separation divides the globe into an upper and a lower half, (2) the vertical plane divides it into an outer and an inner half, and (3) the equatorial plane

divides it into an anterior and a posterior half. The horizontal and vertical lines of separation of the retina intersect in the fovea centralis, and divide the retina into four quadrants. It is necessary to call attention to the *positions of the eyeball,* which are four in number: (1) the anatomic position of rest; (2) the functional position of rest, or primary position; (3) the secondary positions, due to movements from the primary position; and (4) the tertiary positions.

The *anatomic position of rest* obtains in the eyes of the dead and in sleep. It is due to the form of the orbit, the length and insertion of the optic nerve, and the length of the muscles when not innervated. It is usually widely divergent, rarely parallel, and almost never convergent. Since the functions of the eyes cannot be performed in this position (because deviation would cause diplopia), it follows that, on awakening, the eyes unconsciously assume parallelism, and thus are in the *functional position of rest.* In this position the eyes may be said to be held taut by a delicately balanced muscular harness. The lines of vision are parallel and the visual planes are horizontal. This position is called "functional," because the use of the eyes produces it, and it is called the "position of rest," for the reason that normal eyes naturally assume this position without conscious innervation or strain. The *secondary positions* result when movements from the functional position of rest occur. Secondary positions are of two kinds: *(a)* the visual lines are parallel, but are directed upward or downward; and *(b)* the lines of vision converge or diverge. The amount of deviation is expressed by the angle of lateral rotation. From the primary position (functional position of rest) the eye can be rotated 42 degrees outward, 45 degrees inward, 34 degrees upward, and 57 degrees downward. The *tertiary positions* are caused by movements of the eye in which the lines of vision are convergent and at the same time are directed upward or downward.

The movements of the eyes in their various directions are accomplished by the extra-ocular muscles in accordance with *Listing's law,* viz.: when moved from the functional position of rest, the angle of rotation in the second position is the same as if the eye were turned about a fixed axis perpendicular to the first and second positions of the line of vision. Physiologists have shown that in moving from the primary position into any other there is practically no wheel movement of the eyeball around the visual axis, although Javal has demonstrated a slight rotation in this direction. This fact was verified by Helmholtz. The movements of the eyes are direct and oblique, and are the result of the action of muscles as follows:—

Direct movements	Nasally	Internal rectus.
	Temporally	External rectus.
	Upward	Superior rectus and inferior oblique.
	Downward	Inferior rectus and superior oblique.

Oblique movements	Upward and nasally	Superior and internal recti and inferior oblique.
	Downward and nasally	Inferior and internal recti and superior oblique.
	Upward and temporally	Superior and external recti and inferior oblique.
	Downward and temporally	Inferior and external recti and superior oblique.

Protrusion and Retraction Movements.—*The eyes protrude* when the return of venous blood is interfered with, as in hanging; in contraction of the unstriped muscular fibres of Tenon's capsule, innervated by the cervical sympathetic; in forced opening of the lids, which diminishes pressure on the globe; and in excessive action of the oblique muscles. *The eyes retract* when the lids are closed forcibly, in atrophy of the orbital tissues, and after excision of the superior cervical ganglion of the sympathetic

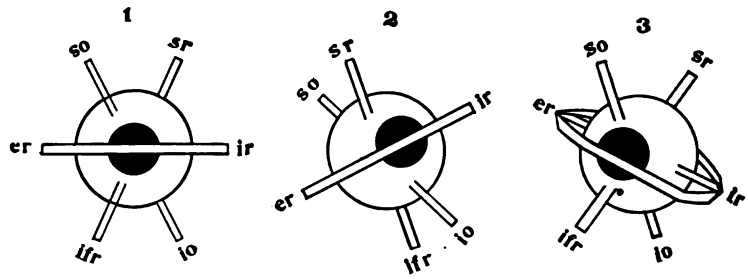


Fig 69.—Diagram to show (1) the primary position of the right eye; (2) the eye turned upward and inward, and (3) downward and outward.

er, External rectus. *ir*, Internal rectus. *so*, *io*, Superior and inferior obliques. *sr*, *ifr*, Superior and inferior recti.

nerve. It will be observed that the conditions under which the eyeball moves antero-posteriorly are usually pathologic.

Relative Ocular Movements.—Normally the eyes move in unison because of the necessity of having an image fixed on each fovea. If we look at an object at a certain distance, and then at another at the same distance, the eyes make *associated movements,* both turning up or down, to the right or to the left, as required, one eye moving as much as the other. When the objects are both situated in the same median plane, in order to fix from the distant to the nearer one, a *movement of convergence* is required, both eyes turning inward to the same degree. When two objects are in different directions, the second nearer than the first, a combination of associated and convergent movements is necessary. A movement of one eye causes the other to move also, or at least to attempt to move. The impulse to binocular single vision disappears during sleep, when the eyes assume the anatomic position of rest. When struggling against sleep, or when suddenly awakened, a momentary diplopia is noticed.

Blindness During Ocular Movements.—Dodge has shown that in the fractional part of a second in which the eyes move the individual is practically blind. The proof of this is that one is never able to see the movement of his own eyes in a mirror.

Binocular Single Vision.—Binocular vision means the union in one single impression of images received simultaneously on both retinæ. Under normal conditions an image of every object looked at is impressed on each retina. The question arises: how is it that these images are blended so that the observer is conscious of but one object? Two chief theories have been proposed for the solution of the problem: (1) the theory of identical points, and (2) the theory of projection. The latter theory assumes that the retino-cerebral apparatus, by a process of mental projection of the image into space, has the power of appreciating the shape and size of an image, as well as the direction of the rays of light which form it. While the limits of this treatise will not permit an extended discussion of the subject of binocular single vision, it will be necessary to consider the first theory more extensively.

The theory of identical points assumes a correspondence of each point of one retina to a similarly situated point on the retina of the other eye. When the eyes are directed toward a far-distant object, the visual axes being then parallel, a correspondence actually exists; but when the visual axes converge the points do not converge. Furthermore, a part of each retina has no corresponding points in the other. This is due to the fact that the actual centre of the retina is not at the fovea centralis, but lies nearer the nasal side. So long as the images of a point are within the horopteric circle they fall on corresponding parts of the retinæ. Images of a point outside this circle do not fall on corresponding points.

This circle, the horopter of Joannes Müller, may be described as being, in the simplest form, a circle which embraces the centre of rotation of each eye and the apex of the point of fixation of the visual lines. Thus, in Fig. 70 it is a simple problem in geometry to demonstrate that images of any point lying within the horopteric circle fall on corresponding retinal points. Thus, the images of *B* will fall on such points. For, if *C* and *D* are nodal points of the two eyes, and *F* and *H* are the centres of the foveæ, the angles *D-A-C* and *D-B-C*—being angles in the same segment of a circle—are equal. In the triangles *A-I-C* and *B-I-D*, the angles *A-I-C* and *B-I-D*—being opposite angles—are also equal. Therefore, the angles *A-I-C* and *B-I-D* are equal; and the angles *E-C-F* and *G-D-H* are equal. Now, if the left eye be placed on the right, so that *C-F* corresponds with *D-H*, *G* must correspond with *E*.

Since the doctrine of identical points is true for only some visual acts, an attempt must be made to explain binocular single vision without the horopter. Such objects as are situated outside the horopter are seen double, but it is possible to obtain only a single cerebral impression of them. Thus, the expert ophthalmoscopist keeps both eyes open while

examining the fundus. The image seen by one eye in this case is ignored. Under some circumstances a new mental picture of two combined dissimilar retinal impressions is made. Thus, ideas of solidity and depth are obtained in binocular vision by the mental combination of dissimilar retinal impressions, as in using the stereoscope. This is an instrument by means of which two somewhat similar pictures, drawn in perspective, are superimposed so that they appear single. One reason why non-identical points yield good vision is that vision becomes less distinct as we pass from the centre of the retina, and the observer learns to neglect the blurred peripheric images while giving attention to those formed on the fovea.

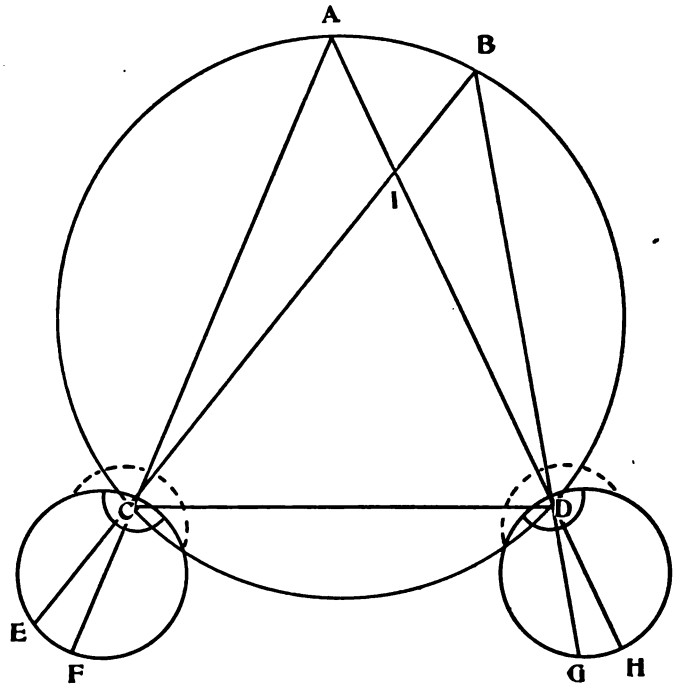


Fig. 70.—The horopteric circle of Joannes Müller.

Stereoscopic Vision.—This is another name for binocular vision, or the sense of depth. The law of identical points was supposed to be absolute up to the time that Wheatstone constructed his stereoscope. This shows that the perception of depth is caused by a slight non-identity of the two retinal images. An object appears to us to be solid when each eye views it from a different point, as in normal vision. Thus, let the observer look at a house. One eye sees more of one side of the house than the other, and *vice versa*. Thus, each eye has its own picture of the house, and the simultaneous use of both eyes gives us a correct idea of the third dimension. It is the unlikeness of the two pictures which gives the idea of depth. The stereoscope is an instrument for viewing two similar pic-

tures which are made to overlap, giving the appearance of solidity and depth. The stereoscopic field of vision, or region in which the visual fields of the two eyes overlap, subtends an angle of about 90 degrees.

Binocular vision is acquired during the first months of life. In babes the eyes wander aimlessly; soon, however, the child learns to fix objects, and this marks an important event in its ocular and cerebral development.

FUNCTIONS OF THE RETINA.

The part of the retina essential to vision is the layer of rods and cones. It cannot be said, however, that the retina sees: what it does is to receive stimuli of light. These are transmitted via the optic nerves and tracts to the brain, where certain changes occur, the nature and site of which are

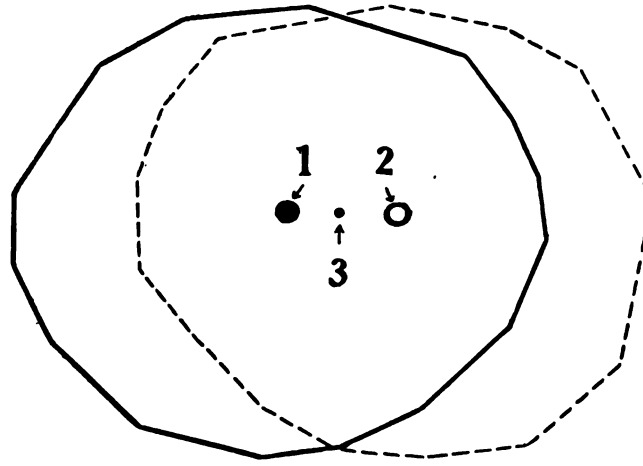


Fig. 71.—Diagram of the field of binocular vision.

The field for the left eye is bounded by the black line; that for the right eye is bounded by the interrupted line. *1, 2*, Physiologic scotomata. *3*. Fixation-point.

entirely unknown. The resulting sensation of light is elaborated into perception of external objects and judgment of the relation which these objects bear to each other and to the observer. Stereoscopic vision having been mentioned already, it will be necessary to consider now the field of vision, the blind spot in the field, the region of most distinct vision, the duration of visual sensations, the inversion of the image, color-vision, and other topics naturally included under the head of the functions of the retina.

The Field of Vision.—Every eye possesses a certain amount of indirect, or peripheral, vision, which when outlined upon a surface is called the field of vision. The recording of this peripheral power of the retina is called measuring the field of vision. The methods used in its determination are discussed in the succeeding chapter. It is sufficient in this place to state

that the field varies according to the facial configuration of the individual and the test-color used, being more extensive on the temporal side than on the nasal, superior or inferior; and more extensive for white than for colors. The position of the eye in the orbit, the prominence of the bones at the base of the orbit, the length of the optical axis, and the size of the pupil are all factors influencing the form of the visual field. The dimensions of the normal field for white and colors are:—

	White.	Blue.	Red.	Green.
Temporally	90°	80°	65°	50°
Nasally	60°	55°	50°	40°
Superiorly	45°	40°	35°	30°
Inferiorly	70°	60°	45°	35°

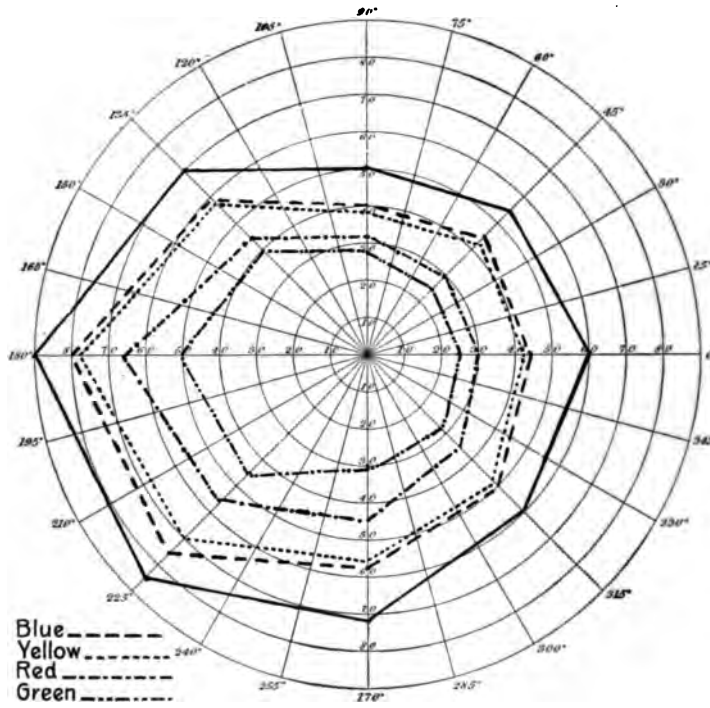


Fig. 72.—Diagram of the normal visual field for white and colors. (Jennings.)

The outer, continuous line indicates the limit of the field for white, and the broken lines indicate the limits of the color-fields.

The Blind Spot.—At the point where the optic nerve enters the globe there are no retinal elements; hence this part of the visual field is blind. A demonstration of the blind spot can be made by a simple experiment. Let the observer close the left eye and with the right look intently at the cross in Fig. 73, the book being held at about twelve inches' distance. At or near this point the black circle will disappear. At that instant rays of

light from the circle strike the head of the optic nerve and the black spot becomes imperceptible. The form of the blind spot as determined by perimetry is elliptical. Its existence proves that the fibres of the optic nerve are insensible to light, which can stimulate them only by means of the retina.

The Macula Lutea.—This is the region of most distinct vision, and it is here that the end-organs of the optic nerve are most highly developed. In daily use of our eyes we unconsciously look directly at objects. Then the image is received on the maculæ and perception reaches its maximum acuity. Any pathologic change in the macular region diminishes visual acuity to a marked extent. Rays of light reaching any other part of the retina result in the formation of an indistinct image.

Direct and Indirect Vision.—We speak of *direct vision* when the image falls on the centre of the macula. *Indirect vision* occurs when the rays from an object fall on the peripheral part of the retina. Indirect vision—although much less acute than direct—is of great assistance in diminishing movements, changes, or intermission of visual impressions (Exner). Direct vision is tested by means of types; and indirect vision is tested by the use of the perimeter.

Fig. 73.—Diagram to show the blind spot in the visual field.

The Rods and Cones in Vision.—The statement has been made that the part of the retina essential to vision is the layer of rods and cones. That this is true is shown by the figures of Purkinje. It was this physiologist who first showed that the shadows of the blood-vessels of the retina can be seen under favorable conditions, although they are ignored in ordinary vision. If, by oblique illumination, light is concentrated far back upon the sclerotic, the eye being directed to a dark background, images of the retinal vessels will be seen as shadows. Since the vessels lie in the anterior portion of the retina, it must follow that in Purkinje's experiment their images are perceived by some part of the retina behind the vascular layer. It has been shown by mathematical calculations, based on the movement of shadows following the movements of the illumination in the experiment mentioned, that the visual impulses originate in the outermost part of the retina: *i.e.*, in the layer of rods and cones. Another argument in favor of the rods and cones as the sensitive part of the retina is furnished by these facts: Since the sensitive part is shown by Purkinje's experiment to be behind the vascular layer, it must be admitted that the part of the retina essential to vision is either the layer of rods and cones, the external nuclear layer, or the pigmented epithelium. The layer of

pigment-cells is excluded, because albinos, both human beings and animals, in whom this layer is absent, possess vision. The external nuclear layer can be excluded for the reason that it is practically absent in the part of the retina where vision is most acute.

Consideration of the nature of the change in the layer of rods and cones which is necessary to the transmission of stimuli is pure speculation. Nevertheless the statements to follow may be assumed to represent correctly the present state of our knowledge of visual sensation. The stimulus which excites vision consists of ether-waves, which are disturbances of definite periodicity in space and time. Variations in the length of waves produce different effects on the retina, and are of importance in exciting a definite series of color-sensations. Physicists tell us that there is no real distinction between heat- and light- waves. The length of wave which excites extreme red is thirty-two millionths of an inch.

The effects produced on sentient cells by light are: (1) a change in color of the purple of the rods, ending in complete bleaching; (2) retraction of the cone, which previously projected into the pigment epithelium; and (3) the generation of an electric current, which travels from the pigment epithelium to the nerve-fibre layer, and is positive.

Many theories have been advanced in explanation of the retinal changes accompanying the visual act, and of these the most plausible is founded on the rediscovery of the visual purple by Boll. This, the photochemical theory, has not cleared the mystery. The limits of this work preclude a discussion of the subject. It is necessary, however, to mention the retinal pictures.

Optograms, which are pictures appearing on the retina after exposure to light, are due to bleaching of the retinal red. They can be obtained by this process: Enucleate the eye of an animal in a dark-room, and keep the eye in a closely covered box. Then direct it toward a window and expose it to bright illumination for a few minutes. The eye is then taken into a dark-room and examined. A picture of the window will be found on the retina, the window-glass appearing light and the sash dark.

Duration of Visual Sensations.—The duration of a visual sensation is always greater than that of the stimulus causing it. However brief the stimulus, the retinal effect lasts about one-eighth of a second. A common experiment in evidence of this proposition is to look at a wheel revolving rapidly. The spokes do not appear as separate radiating lines, but the wheel seems to be one solid mass. This is because each spoke follows another so rapidly that one impression cannot disappear before another is produced. When the stimulus is of some duration, the observer can distinguish between that part of the sensation occurring while light still falls on the eye and that part which remains after the light has ceased to reach the retina. The latter portion is known as an *after-image.* Physiologists classify after-images as positive and negative. The former will generally be of the same color as the stimulus producing them, while the latter, which

are due to fatigue of the stimulated retina, as a rule assume a complementary hue. Thus, if the observer looks for an instant at a very bright light, as at the sun, and closes the eyes, after-images of the luminous body will remain for a few seconds.

Irradiation.—Luminous impressions often are not confined to the retinal elements directly involved, but extend to adjacent elements. This leads to deception as regards the dimensions of objects. Thus, in Fig. 74, the two circles are of the same diameter, yet the white circle on the black ground appears the larger. On prolonged inspection of the objects the deception increases. Irradiation is to be explained by the aberration of the dioptric media of the eye. Not all the rays coming from an object are focused on the retina, and thus the image is surrounded by diffusion circles separating it from the unilluminated area.

Relation of Sensation to the Intensity of the Stimulus.—Physiologists, by comparing the visual sensations caused by different degrees of luminosity, have found that, within certain limits, the smallest difference of light which the human eye can appreciate is about $^1/_{100}$ of the total luminosity.

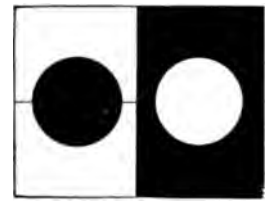


Fig. 74.—Diagram to show irradiation.

Time Necessary for Excitation of the Retina by Light. — This is exceedingly small. It has been computed that light thrown from a rotating mirror stimulates the eye when acting for only $^1/_{8\,000\,000}$ part of a second.

Clearness of Vision is dependent on the space between the retinal cones in the macula lutea. To be clearly seen an object must practically subtend an arc of 60 or 70 seconds in the field of vision. The image of such an object produces an image of about $^1/_{12\,000}$ of an inch in the retina, and this is approximately the distance between the cones.

Inversion of the Image.—It is known from the laws of optics, and from experiments, that the image formed upon the retina is inverted; yet it is perceived as an upright object. Why is it that objects are seen erect when their images are inverted? Without discussing the question extensively it is sufficient to say that it is the result of lifelong habit. A person born blind, and remaining in that condition for many years, as a result of a successful operation, has his sight given him, but is unable by this sense alone to tell the difference between a cube or a sphere, a dog or a cat. All objects appear to him as flat. Soon, however, aided by the sense of touch, he appreciates form, and guided by experience he soon learns to view the external world normally. By touch the individual corrects his

mental impression, and soon the brain learns to make the correction independently. In discussing this question Helmholtz said that, since "our natural consciousness is completely ignorant, even of the existence of the retina and of the formation of images, how should it know anything of the position of images formed upon it?"

The Visual Centre.—The visual fibres in man pass from the optic tract to the external geniculate body and pulvinar. Thence the visual impulse traverses fibres which pass through the posterior end of the internal capsule and run, as the optic radiations of Gratiolet, to the cerebral cortex in and around the calcarine fissure (Munk's visual centre). It is not known whether the nerve-cells of this area are the seat of conscious impressions or are merely substations on the way to higher centres. Whether the same brain-cell is capable of appreciation of differences in color as well as in the amount of light-effect is also unknown.

Color-perception.—When a beam of sunlight is passed through a prism it is decomposed into a series of colored rays called the solar spectrum. These spectral colors are red, orange, yellow, green, blue, indigo, and violet.

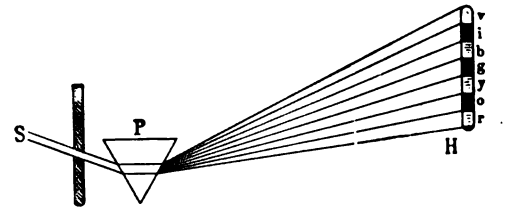


Fig. 75.—Diagram of the decomposition of solar light into the spectral colors. (JENNINGS.)

They are called simple colors because they cannot be further decomposed. The shape of the spectrum is elongated for the reason that the violet rays are more refrangible than the red ones. The spectral colors are said to consist objectively of rapid transverse vibrations of the all-pervading ether, from four hundred millions of millions per second for red to about seven hundred and sixty millions of millions for violet. Subjectively they are sensations caused by the impact of ether vibrations upon the retina. White light, having been decomposed into the colors named above, can be recomposed by means of a biconvex lens. Another method of making white light from a combination of the spectral colors can be demonstrated by means of Newton's disc, which is a piece of cardboard on which five spectral colors are pasted in the shape of triangles radiating from the centre, which is black. By rapidly rotating the disc the observer sees a grayish-white color. Since the duration of retinal impressions is greater than the stimulus causing them, a mixture of the colors occurs, with grayish white as a result.

Colors are distinguished according to (1) their hue, (2) their purity, and (3) their brightness. Tone, or hue, depends on the wave-length: *i.e.*,

the position of the color in the spectrum. Purity, or saturation, depends on the white which is found in nearly all colors except those of the spectrum: the less the amount of white, the greater the purity of the color. The intensity, brightness, or shade depends on the intensity of the illumination. Complementary colors, those which when mixed produce white, are as follows:—

Red and greenish blue.	Yellow and indigo-blue.
Orange and cyanogen-blue.	Greenish yellow and violet.

Red is complementary to green-blue, green to purple, yellow to indigo, and orange to blue.

These are spectrum-colors, and their fusion gives a result different from that obtained by the mixing of pigments of the same colors. Thus, the yellow and indigo-blue of the spectrum make white, while the same pigment-colors produce green.

Black is a sensation which corresponds to a state of rest of the eye. There are no completely black objects in nature: those which seem blackest reflect a considerable quantity of light.

Normal Variation in Color-perception.—While it has long been known that certain individuals cannot distinguish between red and green, it has been supposed until recently that with the exception of these color-blind persons the rest of the human race could distinguish colors accurately. The recent experiments of Rood show that there is as much variation in all people in the power of accurate color-perception as in visual acuity. His observations were carried out by means of the flicker photometer, for the purpose of comparing his own color-vision with that of others. He found that not a single person agreed with him, and no two agreed with each other. In further study of the subject he compared his own with the color-perception of eleven persons. It was found that they could be divided into two classes according to their perception of green. The average color-vision of the eleven was taken as the normal standard, and the divergence of each person from this standard was then calculated.

Theories of Color-perception.—These are numerous. No two of them agree and not one is entirely satisfactory. The whole question resolves itself into the fact that there is a pair of receptive elements with adaptive apparatuses that are capable of receiving light-rays and transferring them into energies that are carried to the occipital cortex, in which situation they are gotten ready for perception.

The Ocular Adnexa.—The eyelids afford protection to the globe. The eye is opened by the action of the levator of the upper lid (which is innervated by the third nerve) assisted by depression of the lower eyelid. The eye is closed by contraction of the orbicularis palpebrarum muscle, which is supplied by the facial nerve. Closure is generally a reflex act, but is under voluntary control. The reflex act of winking is under the control of afferent impulses carried by the ophthalmic division of the fifth

nerve and efferent impulses transmitted by the facial. Since the levator palpebræ superioris muscle sends a slip to the superior rectus, contraction of the latter not only raises the visual axis, but also elevates the upper eyelid to a slight extent. The inferior rectus likewise depresses the visual axis and lower eyelid.

The conjunctiva is the membranous covering of the eyelids and globe. It is constantly kept moist by the secretion of the lacrimal gland of the orbit and the accessory glands found in the lids. The fluids thus formed ordinarily disappear partly by evaporation, partly by way of the drainage apparatus. The lacrimal fluid is slightly alkaline in reaction, containing about 1 per cent. of solids, among which sodium chlorid is noticeable. Excessive lacrimal secretion, or the production of tears, is a reflex act occurring in response to irritation applied to the conjunctiva, cornea, skin of the face, nasal mucous membrane, tongue, buccal mucous membrane, or any peripheral irritation or lesion causing pain. It also occurs in response to the emotions and as a result of stimulation of the retina by strong light. Section of the cervical part of the sympathetic nerve produces lacrimation. The sensory part of the fifth is the afferent nerve. The efferent fibres are found in the lacrimal and orbital branches of the same nerve.

The flow of the lacrimal secretion into the canaliculi, sac, and lacrimo-nasal duct occurs in response to mechanical force, in which the chief factors are capillarity, gravity, and the movements of the lids. The act of winking rapidly prevents an overflow of tears. Henke has compared this action to that of a suction-pressure pump. The generally accepted view is that muscular action expands the lacrimal sac, into which the tears are drawn. The sac is emptied by the passive contraction of its elastic fibres.

Lacrimal secretion is generally absent from the eyes of the newborn, and is not noticed until about the second month of extra-uterine existence. The naso-lacrimal duct, often imperfect at birth, becomes patent about the same time.

CHAPTER IV.

EXAMINATION OF THE EYE.

EXAMINATION of the eye includes ordinary inspection, inspection by the aid of lenses and instruments of precision, ophthalmoscopy, and functional testing. Inspection and ophthalmoscopy are fortunately independent of the answers of the patient; the functional tests depend entirely on his replies. All of the methods are of value. In addition to their use the surgeon should secure a complete history of the case and should make a record of the same. Observance of this rule will not only save much time if the patient should return at a later period, but will furnish data of value to the profession. Furthermore, the possession of a case-history may protect the surgeon from a suit for alleged malpractice. A systematic plan of examination should be followed, the record being placed in book or card form. For this purpose the following order may be used:—

Name..... Residence..... Age..... Occupation.....

Sex..... Color..... Married, single, or widowed.....

General and ocular diseases of parents.....

Personal history of patient: constitutional diseases, acquired or hereditary; injuries .....

Personal habits of patient: alcohol, tobacco, drugs, sexual relations .....

.....

Date and symptoms of present trouble: pain in or around the eyes, symptoms caused by use of the eyes, etc.....

.....

External examination: inspection of both eyes shows.....

Inspection of right eye: lids, conjunctiva, cornea, tension, pupil, etc. .....

Inspection of left eye: lids, conjunctiva, cornea, tension, pupil, etc. .....

Vision of right eye = ...; improved by convex or concave sphere or cylinder

.....

Vision of left eye = ...; improved by convex or concave sphere or cylinder

.....

Examination of right eye with ophthalmometer shows.....

Examination of left eye with ophthalmometer shows.....

Muscle-balance measured by Maddox rod, phorometer, etc., shows { Right..... Left.....

Ophthalmoscopic examination shows { Right..... Left.....

Record of the visual fields { Right..... Left.....

Examination of color-sense { Right..... Left.....

Examination of refraction after the use of a mydriatic for ..... days { Right..... Left..... { Homatropin. Scopolamin. Atropin.

Examination by retinoscopy { Right..... Left.....

X-ray examination by Dr.....

Diagnosis and prognosis.....

Local remedies ordered applied.....

Internal remedies prescribed.....

Glasses and frames prescribed { Right..... Left.....

Patient is to return.....

Patient referred by Dr.....

Remarks .....

EXTERNAL EXAMINATION.

Having obtained the history of the case, attention is naturally first directed to the two eyes to determine any difference in appearance or lack of symmetry. Such marked difference as the absence of one eye, a turning of the globe, the presence of a swelling of an inflammatory nature, or the existence of a tumor or ulcer will immediately be noticed. In many cases, however, the departure from the normal is evidenced by slight changes, which call for minute examination. First in order will be *inspection of the eyelashes.* Notice should be taken whether the lashes are present in normal number, evenly distributed along the margin of the lid, or if certain spots seem to show too few cilia. Also it should be noted if the cilia are properly directed. If they turn toward the globe, note whether they are improperly directed (trichiasis) or if they are present in two or more rows of misplaced cilia (distichiasis). The lashes may be normally

distributed and directed; their ends do not taper to a fine point, but appear as stubs. In this case, ask if the patient has trimmed the lashes or has had them singed either intentionally or by accident. Search should be made for small, white, downy hairs, which may grow from any part of the lid-margin and are often found at the inner canthus, where they rub the globe or caruncle with every wink. Short, thick, black hairs, known by the laity as "wild hairs," often cause much irritation. About the roots of the cilia parasites (phthiriasis oculorum) may be found. The matting together of the lashes with a moist substance will at once suggest the presence of some form of conjunctivitis.

Eyelids.—Any change in color, size, contour, or motility of the lids should be observed. The lid is subject to eczema, xanthelasma, and other cutaneous diseases. A localized inflammatory swelling of the lid will suggest trauma, hordeolum, erysipelas, erythema, sarcoma, an inflamed chalazion, or acute circumscribed edema. A local non-inflammatory swelling may be due to a dislocated lacrimal gland or to a chalazion. In the latter case a hard, round body like a shot will be felt. A local non-inflammatory swelling may be due to nephritis. A general inflammatory swelling of the lid is met with in trauma, gonorrheal conjunctivitis, and erysipelas. A general non-inflammatory swelling may be caused by emphysema, in which case palpation will elicit the characteristic crepitation; or it may come from the accidental discharge of a solution which has been thrown into the cellular tissue while injecting the naso-lacrimal duct.

An ulcer of the lid may be due to syphilis (chancre), in which case the pre-auricular gland will be enlarged, or to a malignant growth (epithelioma).

Nevi and varicose veins are sometimes seen in the lids.

As regards motility, it is noticed in ptosis that the lid can be raised only by calling the occipito-frontalis muscle into action.

In exophthalmic goitre the upper lid follows the globe slowly when the patient is told to look down (von Graefe's sign) or is retracted (Dalrymple's sign); or the act of winking may be done at long intervals (Stellwag's sign). Drooping of the lid, aside from inflammatory thickening, is found in paralysis of the cervical portion of the sympathetic nerve, which supplies the fibres of Müller's muscle. Vertical narrowing of the palpebral fissure exists in enophthalmos. Imperfect closure of the lids is found in paralysis of the seventh nerve. The levator palpebræ superioris muscle may act only in conjunction with another muscle or group of muscles. In these instances the patient always shows slight congenital ptosis. He should be told to look downward, opening his mouth widely and moving the jaw from side to side. It will be found that the levator then draws the lid well above the cornea. In some cases the levators act only in conjunction with the internal recti, convergence causing the upper lids to be retracted.

The contour of the lid may be changed by scars from wounds, ulcera-

tion, or burns. The finger should be passed along the lower part of the anterior surface of the upper and corresponding portion of the lower lid, to feel for thickened areas. If present, they will suggest tarsitis or occlusion of the Meibomian ducts. The position of the edges of the lids may be changed. A turning inward, entropion, will cause the cilia to brush against the cornea. A turning outward, ectropion, will expose the inflamed and thickened conjunctiva and cause a flow of tears on to the cheek. Attention should be given to the shape of the lid-margin. Normally the margin is rounded on the skin side, while the side toward the globe forms a right angle. In trachoma the posterior lip is worn away. Having inspected the external surfaces and margins, the examiner should proceed to the next step.

EVERSION OF THE LIDS may appear to be a simple matter, but under pathologic conditions eyes may be injured by unskillful manipulation. Slight force expended in the proper way will enable the examiner to evert the eyelid without pressing on the globe. The procedure differs according to the age of the patient. In examining an adult the surgeon should stand

Fig. 76.—Eversion of the upper eyelid.

behind a chair on which the patient sits. The patient should look downward. The examiner, grasping the eyelashes of the upper lid between the thumb and index finger of the left hand, applies the index finger of the right hand to the outer surface of the lid at a point midway between the lid-margin and the margin of the orbit. The lid is to be gently lifted over the fulcrum thus made by the right index finger. So long as the patient looks downward the turning of the lid is generally easily accomplished.

To evert the lower lid of the adult the examiner should be seated facing the patient, who is to look upward. At the same time the examiner pulls the lid downward, the eversion being done by means of the thumb applied to the cutaneous side of the lid.

In eversion of the lids of a child the surgeon is to sit facing the nurse, who, grasping the child's hands and legs, places the patient in a horizontal position, the head being toward the examiner. Seizing the child's head firmly between his knees, the surgeon is free to use both hands. Since

children frequently are terrified and struggle, care must be taken to avoid pressure on the globe. Often, by waiting until the child becomes tired, the upper lid can be everted by simply making traction with the thumb placed at the orbital margin; or the lid may be everted over a match, which is used as a fulcrum. The eversion of the lower lid in the child is easily accomplished.

The lids being everted,—first the upper, next the lower,—search should be made for pathologic conditions, such as conjunctivitis, fissures at the outer canthus, deformity of the upper tarsal plate, areas of ulceration, or patches of atrophied conjunctiva forming guy-ropes binding the lid to the globe. At the same time the condition of the cornea can be observed. Search for foreign bodies should not be omitted. By the methods described above the lower conjunctival fornix can be thoroughly explored, but not the upper. To explore this requires another procedure known as

Secondary Eversion of the Upper Lid.—Having turned the upper lid and completed its inspection, the surgeon should retain the lid in its

Fig. 77.—Examination of the eye of a child.

everted position and pass beneath it, and into the upper fornix, a Noyes retractor (Fig. 78). This instrument is used to expose the entire upper fornix, which is a favorite location for trachoma and foreign bodies. Papillary tumors of the conjunctiva occasionally grow from this part, owing to the irritation caused by the lodgment of a foreign body. To render this procedure painless a few drops of holocain (1-per-cent. strength) can be dropped into the eye. Only rarely does it occur that spasmodic closure of the lids cannot be overcome by the means just described. If so, a general anesthetic will be necessary. While the lids are everted any required application should be made.

Ocular Conjunctiva.—At the same time that the palpebral conjunctiva is examined the condition of the ocular portion of this membrane should be determined. If undue redness is present, notice should be taken

whether it is greatest posteriorly, in the region of the fornices, or anteriorly, around the corneoscleral junction. The presence of trachoma bodies or foreign substances will be noticed. Ulcerations of the ocular conjunctiva occur as broad, flat losses of tissue, while in conjunctival tuberculosis the ulceration presents a thickened, irregular, mammillated appearance. Broad, hard patches of infiltration may be due to the initial lesion of syphilis or to a beginning epithelioma. Benign tumors occur in or immediately beneath the ocular conjunctiva. Localized patches of hyperemia, of a bright-red color, will suggest episcleritis. Invasion of the cornea by con-

Fig. 78.—Noyes's retractor.

junctival vessels in the form of pannus is very common. A deep-pink zone of redness, involving vessels beneath the conjunctiva, suggests a deep-seated inflammation of the eye, involving particularly the uveal tract. To determine whether the redness is in or beneath the conjunctiva the observer needs only to move the eyelid over the eyeball; and since the ocular conjunctiva is loosely attached to the globe, movement of this membrane can be readily caused. If the redness moves with the conjunctiva, it is due to conjunctival hyperemia; if not, it is deeply seated. Both forms of hyperemia may exist at the same time in the same eye. The deep-seated zone of redness is greatest immediately around the corneoscleral junction.

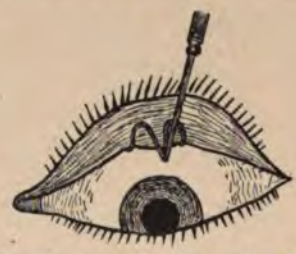


Fig. 79.—Position for secondary eversion of the upper lid.

Nettleship distinguishes three systems in the blood-supply of the anterior part of the eye. These vessels, while scarcely to be seen in health, are prominent in disease.

System I.—The posterior conjunctival arteries and veins (*A*, Fig. 80). Hyperemia of these causes a bright-red color, which moves with the conjunctiva. It is associated with a muco-purulent discharge and indicates conjunctivitis. The greatest redness is in the fornices.

System II.—The anterior ciliary vessels *(B)*, which supply the sclera, iris, and ciliary body by means of perforating branches, while the non-perforating vessels supply the episcleral tissue. Congestion of these vessels

produces a pinkish zone of circumcorneal congestion which does not move with the conjunctiva. Hyperemia of this system is present in iritis and cyclitis. Venous congestion of this region is often present in glaucoma as a dark, violet-colored zone.

System III.—The anterior conjunctival and ciliary vessels. These are minute vessels which form loops in the peripheral part of the cornea. Hyperemia of these causes a bright-red discoloration around and partly in the cornea, and is typically seen in the early stages of interstitial keratitis.

Lacrimal Apparatus.—The examiner should notice whether the puncta are directed properly, so as to take up the flow of tears. Often they are everted, or they may be directed properly and be closed; or eye-

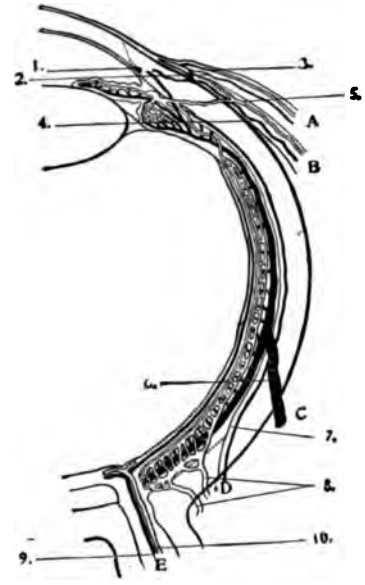


Fig. 80.—Diagram to show the vascular systems of the eye.

A, Conjunctival vessels. *B*, Anterior ciliary vessels. *C*, Vena vorticosa. *D*, Posterior ciliary arteries. *E*, Central retinal vessels.

lashes or other foreign bodies may be engaged in a canaliculus and protrude from the punctum. Normally the punctum cannot be seen except the lid be lifted away from the globe.

The condition of the tear-sac can be determined by palpation. If pressure on it causes the discharge of mucus or pus into the nose below, or, as is more common, into the conjunctival sac above, the sac is diseased. If there is any doubt as to the patency of the drainage apparatus, a few drops of warm boric acid solution should be injected from a hypodermic syringe armed with a blunt, hollow needle, which is placed in the lower canaliculus. If the fluid runs freely into the nose the canals are open. If the fluid returns into the conjunctiva via the upper canaliculus, there is either obstruction or stricture. While this statement is true of most

cases, mention must be made of the fact that a growth of leptothrix occurring in the lower canaliculus has been known to permit the discharge of fluid from a syringe, while the mass caused epiphora, enlargement of the canaliculus, and practically lacrimal obstruction.

Another method of determining the patency of the lacrimal passages is to place a few drops of a 2-per-cent. strength fluorescin solution in the conjunctiva. After waiting a minute or two the patient is told to use his handkerchief. The presence of the fluorescin stain will show that the passages are open.

While in nervous subjects syringing of the naso-lacrimal apparatus should be preceded by the application of a local anesthetic to the conjunctiva, the author does not favor the injection of a cocain solution when the object is to determine the patency of the canal. The constricting effect of the cocain on the mucous membrane may lead to wrong deductions.

The injection of a solution (1 to 10) of suprarenal extract into the lacrimal sac will often enable the surgeon to distinguish between simple inflammatory thickening of the drainage apparatus and true stricture. If the former condition exists, the apparatus soon becomes patent; if the latter, the epiphora continues (Bates).

The accessory lacrimal gland may be seen by drawing the upper lid well away from the globe and at the same time having the patient look downward. The gland proper cannot be seen or felt under normal conditions.

The Caruncle is sometimes deeper on one side than the other, owing to retraction following tenotomy of the internal rectus muscle. Neoplasms sometimes grow from it.

The Sclerotic should be uniformly white in youths and adults and yellowish in very old people. In children it is of a bluish tint. If a bulging of the sclera is seen, it may be simply a staphyloma marking the site of an old injury or idiopathic inflammation, or it may indicate the position of an intra-ocular tumor. If the latter, oblique illumination, the light being concentrated on the protrusion, will not illuminate the interior of the eye. If the protrusion is staphylomatous, light passes freely through the thinned membranes and the interior is illuminated.

The Length of the Eye can be approximately determined by the distance from the height of the scleral curve to the cornea. The patient is directed to turn the eye strongly inward, while the examiner observes it from the outer side. In hypermetropia the equator is nearer and in myopia it is farther from the cornea than in the normal eye.

The Cornea is viewed without turning the lid and normally is transparent. Any pathologic condition is evidenced by a loss of this property. Its diameter can be determined by Priestley Smith's keratometer, which consists of a millimetre scale placed between two plano-convex lenses in the form of an eyeglass having a focal length of ten inches; or it can be measured with the ophthalmometer.

Hemorrhage into the layers of the cornea is a condition rarely found and generally comes from trauma.

Opacities of the cornea often require minute examination for their detection. Daylight is first used, and light is concentrated on the cornea by a double convex lens. The same is done with artificial light. A superficial opacity is called a nebula; a dense one, a leucoma; a vascular one, pannus. Ulceration, interstitial inflammation, abscess, pressure, and foreign bodies will cause opacities. The presence of a vascular keratitis will call for an immediate eversion of the lids and a search for trachoma bodies. The cornea may be clear, but protruding at its centre (keratoconus). It may be immensely enlarged as part of the disease known as hydrophthalmos. Sometimes opacities are present in it at birth.

In examining the cornea the following methods may be employed:—

(A) The patient is placed facing a window, an outline of which will be seen reflected on the cornea. Any irregularity or opacity will cause a distortion of the image of the window.

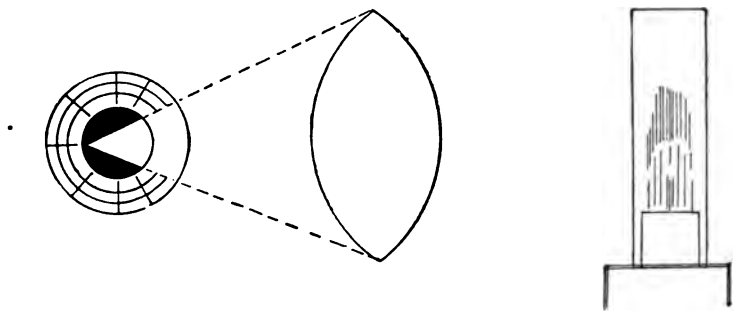


Fig. 81.—Diagram to show focal (or oblique) illumination.

(B) Focal illumination is a valuable method. A double convex lens of two or three inches' focus is used to concentrate the light from a lamp upon the cornea; or the same object can be attained by the use of the candle-lamp of Priestley Smith. Another method is to reflect light from the ordinary head-mirror. Regardless of the method used to concentrate light, it is advisable to view the cornea through a magnifying glass.

(C) The concave retinoscopy mirror of twenty-five centimetres' focus can be used to concentrate daylight or artificial light on to the cornea.

(D) Placido's disc is used. The patient is placed with his back to a window and the examiner reflects light from the disc on to the cornea, the observer looking through a small hole in the centre of the disc. Nebulæ will cause a distortion in the image. The disc can also be used with artificial light.

(E) In case an ulcer or abrasion is small, its outline can be obtained by the use of a solution of fluorescin (2 per cent.), which is dropped on the cornea. Any part of the cornea denuded of its epithelium will be stained a bright-green color, while the normal tissue will be unaffected. The

previous use of cocain increases the staining power of fluorescin. Valuable as is the fluorescin test, it is not infallible. Benson has found that frequently fluorescin would not stain some conditions commonly called ulcers; and, on the other hand, it would stain many corneas where neither ulcers, epithelial abrasions, nor pathologic conditions of the epithelium were discoverable.

(F) The ophthalmometer can be used to determine the radius of curvature of the different meridians of the cornea, as will be explained later.

(G) To determine the sensitiveness of the cornea, which is much reduced in glaucoma and in some diseases of the fifth nerve, take a small wisp or string of absorbent cotton and touch the cornea, taking care not to touch any other part.

Fig. 82.—Placido's disc.

(H) The corneal microscope, or the binocular magnifier of Jackson, can be employed to detect minute changes in the cornea, anterior chamber, and iris. Jackson's instrument is designed to give a true binocular image magnified in all directions. It consists of two tubes with convex lenses at their convergent ends, and prisms at the divergent ends.

The Anterior Chamber should be examined as to its depth, the clearness of its contents, and the possible presence of a foreign body or of an exudation. The depth of the chamber is estimated by observing the relationship of the iris to the anterior surface of the cornea. Normally it averages 2.6 millimetres. Like a brook, when clear, the chamber is deeper than it looks. Normally it is shallower in infants and in the aged than in middle life. Pathologically the anterior chamber may be empty, too shallow, or too deep. When empty, the iris is in contact with the posterior surface of the cornea. A wound or a perforation from an ulcer will then

be found. The chamber may be obliterated in an old case of iridocyclitis, in glaucoma, intra-ocular tumor, or in epibulbar sarcoma and hydrophthalmos.

A shallow chamber occurs in abnormally flat cornea, in traumatic cataract with swelling of the lens, in glaucoma, and in intra-ocular growths. It also is met with in an early stage of senile cataract when the lens is swollen. A deep chamber occurs where the cornea is too protuberant; in case the iris is displaced backward, as in adhesions between the pupillary margin of the iris and the capsule of the lens, the latter being drawn backward by the contraction of old cyclitic membranes. It also is present in serous cyclitis and in aphakia. The chamber may be shallowed or abolished at the periphery and be abnormally deep at the centre, as is sometimes observed in iritis, hydrophthalmos, and in cyclitic eyes. Adhesions between the cornea and iris can be determined either by ordinary inspection or by oblique illumination.

Normally the aqueous humor is invisible. Pathologically it may be mixed with blood, pus, or lymph. Fluids in the chamber gravitate according to the position of the patient. Minute dots on the posterior surface

Fig. 83.—Jackson's binocular magnifier.

of the cornea from a deposition of lymph are found in serous cyclitis. Foreign bodies in the anterior chamber can be seen by oblique illumination. The history of the accident will assist in the determination of their nature. If of iron or steel, the electromagnet will be of assistance in their removal.

The Iris can be readily examined if the cornea and aqueous humor are clear. The color of the iris, its lustre, its movements, and its contour should all be observed. A lack of lustre and a change in color, together with a sluggish or immobile pupil, will indicate iritis. A localized bulging, with surrounding discoloration, may come from a foreign body, an intra-ocular tumor, a parasite, or a gummatous iritis.

Tremulousness of the iris can be elicited by having the patient move the eye rapidly up and down, halting suddenly at the middle line. It indicates a want of support, and is found when the zonula is relaxed or the lens absent, dislocated, or shrunken. The condition can generally be observed easily by daylight. Sometimes it is better seen in a dark-room by oblique illumination.

The Pupil should be carefully studied in all ocular cases, since it furnishes much information not to be obtained elsewhere. The size of the

pupil can be measured by the use of the pupillometer, which the observer holds close to the outer canthus. The disc is turned until an aperture is found corresponding to the size of the pupil. The record is made in millimetres.

Inequality of the pupils rarely occurs in health. It is found where one eye is blind, in disease of the teeth, in traumatism producing minute tears in the sphincter of the iris, in tabes, cerebral syphilis, disseminated sclerosis, paretic dementia, epilepsy, and probably in a few other conditions. Inequality alternating from one side to the other occurs as a premonitory sign of insanity.

Mobility of the iris is determined by placing the patient in front of a window facing the light. He is to look at a distance. The examiner then covers the eyes for a moment; on uncovering, the pupils should be found larger, but should immediately contract. In case the iris is immobile and the tension of the eye is normal, a mydriatic should be used to determine whether adhesions are present. If the pupil oscillates between dilation and contraction, the condition is called *hippus*. This is a rare state, which Knies says "is observed in recovering paralysis of the motor oculi, and is then associated with nystagmus. It is much rarer as an independent condition, and is then found almost always in diseases—such as tabes, multiple sclerosis, etc.—in which there are frequent lesions in the region of the nuclei of the ocular muscles."

CONTRACTION OF THE PUPIL, miosis, may come from (1) irritation or (2) paralysis.

I. Miosis from Irritation is caused by :—

(a) Diffuse inflammatory conditions of the brain and meninges, causing a direct stimulation of the motor oculi nerve.

(b) Tumors near the anterior corpora quadrigemina or near the centre of the third nerve or in its fibres.

(c) In the first stage of apoplexy, in hysteria, and in epilepsy.

(d) In hemorrhage into the pons.

(e) After long-continued near work, causing spasm of the ciliary muscle and sphincter of the iris.

(f) Inflammatory conditions of the anterior portion of the eye (keratitis, iritis, cyclitis, etc.) and foreign bodies in the cornea and conjunctiva.

(g) After the use of eserin, pilocarpin, arecolin, muscarin, nicotin, or opium.

II. Paralytic Miosis, due to the sympathetic nerve, occurs in:—

(a) Injuries, apoplexy, tumors, inflammations of the cervical cord.

(b) Mediastinal tumors and carcinoma of the esophagus.

(c) Paralysis of the sympathetic nerve, and after excision of the superior cervical ganglion.

DILATION OF THE PUPIL, known as mydriasis, can be divided into two types: (1) paralytic and (2) spastic.

I. Paralytic Dilation, dependent on the oculomotor nerve, occurs in:—

(a) Hemorrhage or tumor in the floor of the aqueduct of Sylvius.

(b) In diseases which affect the fibres of the motor oculi nerve anywhere in their course (*e.g.*, sinus-thrombosis and glaucoma).

(c) After the use of atropin, duboisin, daturin, hyoscyamin, hyoscin, or homatropin.

(d) In crushing of the eyeball.

II. Spastic Dilation, dependent on the sympathetic, is found in:—

(a) Fright.

(b) Accumulation of CO_2 in the blood.

(c) In the fully-developed epileptic and eclamptic attack.

(d) Tumors and inflammations of the spinal cord (*e.g.*, in the early stage of tabes).

(e) Reflex action from the presence of worms in the intestine; in lead and biliary colic.

(f) In tumors of the neck.

(g) Melancholia and mania.

(h) After the use of cocain.

(i) In labor and all painful irritations of any part of the sympathetic, such as renal colic, etc.

Facts Concerning Pupillary Reaction.—In miosis due to irritation, light, accommodation, convergence, and eserin will cause still greater contraction; atropin will produce dilation.

In miosis arising from paralysis, light, accommodation, convergence, and eserin will cause contraction; atropin has but little effect.

In paralytic mydriasis there is no reaction with light, accommodation, or convergence; eserin acts but feebly.

In spastic mydriasis, light, accommodation, convergence, and eserin will cause contraction.

Abnormal Varieties of Pupillary Reaction. — These are the Robertson phenomenon, the hemianopic iris-inaction of Wernicke, the cortical reflex of Haab, the skin reflex, and Gifford's contraction.

The Robertson Symptom is this: the pupils contract on convergence or accommodation, but either do not react to light or do so feebly. This sign is brought out by first finding (by covering and uncovering the eyes) that the light-reflex is absent or feeble; next, the patient is told to look at an object placed at ten inches' distance from the eyes. The pupil will be found to contract. It occurs frequently in tabes and general paralysis (two diseases due largely, if not entirely, to syphilis), and has been observed in senile dementia, multiple sclerosis, cerebro-spinal syphilis, epilepsy, lead poisoning, progressive muscular atrophy, aortic aneurism, hemiplegia, nuclear ophthalmoplegia, and chorioiditis.

The Iris-inaction of Wernicke occurs in some cases of hemianopsia, and is valuable in localizing the lesion causing half-sight. A beam of light is thrown on the blind half of the retina. If the pupil does not contract, the lesion is in front of the corpora quadrigemina: *i.e.*, in the optic nerve,

pupil can be measured by the use of the pupillometer, which the observer holds close to the outer canthus. The disc is turned until an aperture is found corresponding to the size of the pupil. The record is made in millimetres.

Inequality of the pupils rarely occurs in health. It is found where one eye is blind, in disease of the teeth, in traumatism producing minute tears in the sphincter of the iris, in tabes, cerebral syphilis, disseminated sclerosis, paretic dementia, epilepsy, and probably in a few other conditions. Inequality alternating from one side to the other occurs as a premonitory sign of insanity.

Mobility of the iris is determined by placing the patient in front of a window facing the light. He is to look at a distance. The examiner then covers the eyes for a moment; on uncovering, the pupils should be found larger, but should immediately contract. In case the iris is immobile and the tension of the eye is normal, a mydriatic should be used to determine whether adhesions are present. If the pupil oscillates between dilation and contraction, the condition is called *hippus*. This is a rare state, which Knies says "is observed in recovering paralysis of the motor oculi, and is then associated with nystagmus. It is much rarer as an independent condition, and is then found almost always in diseases—such as tabes, multiple sclerosis, etc.—in which there are frequent lesions in the region of the nuclei of the ocular muscles."

CONTRACTION OF THE PUPIL, miosis, may come from (1) irritation or (2) paralysis.

I. Miosis from Irritation is caused by :—

(a) Diffuse inflammatory conditions of the brain and meninges, causing a direct stimulation of the motor oculi nerve.

(b) Tumors near the anterior corpora quadrigemina or near the centre of the third nerve or in its fibres.

(c) In the first stage of apoplexy, in hysteria, and in epilepsy.

(d) In hemorrhage into the pons.

(e) After long-continued near work, causing spasm of the ciliary muscle and sphincter of the iris.

(f) Inflammatory conditions of the anterior portion of the eye (keratitis, iritis, cyclitis, etc.) and foreign bodies in the cornea and conjunctiva.

(g) After the use of eserin, pilocarpin, arecolin, muscarin, nicotin, or opium.

II. Paralytic Miosis, due to the sympathetic nerve, occurs in:—

(a) Injuries, apoplexy, tumors, inflammations of the cervical cord.

(b) Mediastinal tumors and carcinoma of the esophagus.

(c) Paralysis of the sympathetic nerve, and after excision of the superior cervical ganglion.

DILATION OF THE PUPIL, known as mydriasis, can be divided into two types: (1) paralytic and (2) spastic.

I. Paralytic Dilation, dependent on the oculomotor nerve, occurs in:—

(a) Hemorrhage or tumor in the floor of the aqueduct of Sylvius.

(b) In diseases which affect the fibres of the motor oculi nerve anywhere in their course (*e.g.*, sinus-thrombosis and glaucoma).

(c) After the use of atropin, duboisin, daturin, hyoscyamin, hyoscin, or homatropin.

(d) In crushing of the eyeball.

II. Spastic Dilation, dependent on the sympathetic, is found in:—

(a) Fright.

(b) Accumulation of CO_2 in the blood.

(c) In the fully-developed epileptic and eclamptic attack.

(d) Tumors and inflammations of the spinal cord (*e.g.*, in the early stage of tabes).

(e) Reflex action from the presence of worms in the intestine; in lead and biliary colic.

(f) In tumors of the neck.

(g) Melancholia and mania.

(h) After the use of cocain.

(i) In labor and all painful irritations of any part of the sympathetic, such as renal colic, etc.

FACTS CONCERNING PUPILLARY REACTION.—In miosis due to irritation, light, accommodation, convergence, and eserin will cause still greater contraction; atropin will produce dilation.

In miosis arising from paralysis, light, accommodation, convergence, and eserin will cause contraction; atropin has but little effect.

In paralytic mydriasis there is no reaction with light, accommodation, or convergence; eserin acts but feebly.

In spastic mydriasis, light, accommodation, convergence, and eserin will cause contraction.

ABNORMAL VARIETIES OF PUPILLARY REACTION. — These are the Robertson phenomenon, the hemianopic iris-inaction of Wernicke, the cortical reflex of Haab, the skin reflex, and Gifford's contraction.

The Robertson Symptom is this: the pupils contract on convergence or accommodation, but either do not react to light or do so feebly. This sign is brought out by first finding (by covering and uncovering the eyes) that the light-reflex is absent or feeble; next, the patient is told to look at an object placed at ten inches' distance from the eyes. The pupil will be found to contract. It occurs frequently in tabes and general paralysis (two diseases due largely, if not entirely, to syphilis), and has been observed in senile dementia, multiple sclerosis, cerebro-spinal syphilis, epilepsy, lead poisoning, progressive muscular atrophy, aortic aneurism, hemiplegia, nuclear ophthalmoplegia, and chorioiditis.

The Iris-inaction of Wernicke occurs in some cases of hemianopsia, and is valuable in localizing the lesion causing half-sight. A beam of light is thrown on the blind half of the retina. If the pupil does not contract, the lesion is in front of the corpora quadrigemina: *i.e.*, in the optic nerve,

chiasma, or optic tract. If the pupil contracts, the lesion is posterior to the corpora quadrigemina: *i.e.*, in the optic radiations or cuneus.

Wernicke's symptom, to be of value, must be carefully sought for. In a dark-room a plane mirror is used to illuminate the eye feebly. With a concave retinoscopy mirror light is to be thrown obliquely into the pupil, first on the blind side, then on the other, and the result is recorded.

The Cortical Reflex of Haab occurs when the patient sits in a dark-room and, on thinking of a bright light, the pupil contracts.

The Skin Reflex is a dilation of the pupil, which occurs when a cutaneous nerve is pinched.

Gifford's Reflex (known also as Galassi's or the Westphal-Piltz reaction) is elicited by holding the lids open by a speculum. When the patient attempts to close the lids the pupil contracts. It is used to determine whether the sphincter of the iris is paralyzed.

The Pupillary Area is to be examined by oblique illumination. Search is to be made for adhesions of the iris to the capsule of the lens, for exudations, and for pigment-spots.

The crystalline lens is examined (1) by oblique illumination; (2) by the ophthalmoscope or preferably by the concave retinoscopy mirror; (3) by illumination through the sclera. The mirror of the ophthalmoscope is generally oblong, while the retinoscopy mirror is round and large (diameter, 1 to 2 inches) and gives a better illumination.

Search is to be made for opacities or foreign bodies. Opacities viewed by oblique illumination appear grayish or whitish on a black background; if seen with a mirror, they are black on the red fundus. Only those opacities can be observed which correspond to the pupillary area. To examine the periphery of the lens the use of a mydriatic will be necessary. In the selection and use of a mydriatic the examiner should be guided by the rules given elsewhere in this chapter.

Foreign bodies often lodge in the lens, where they can sometimes be seen. Generally they are obscured by rapid swelling of the lens-substance, and their presence can be demonstrated by the use of x-ray photography after the method of Sweet or of Davidson. Occasionally oblique illumination will show a rent in the capsule of the lens caused by the foreign body. Small bodies may lodge in the lens and remain for years without giving rise to trouble.

After looking at the lens by the oblique method the observer is to use direct ophthalmoscopy, placing a convex glass of 10 D. strength behind the mirror of the ophthalmoscope. This enlarges the parts. If the lens is dislocated, colobomatous, or if an iridectomy has been made, the observer can see the margin of the lens. Sometimes the suspensory ligament and tips of the ciliary processes are visible.

The Crystalline Lens.—There are two methods of examining the lens: (1) oblique illumination, and (2) the use of the ophthalmoscopic mirror. Opacities in the lens appear grayish when seen by the oblique method; if

observed by transmitted light they look black on a red background. To examine the peripheral parts of the lens the use of a weak mydriatic, such as mydrin, euphthalmin, or homatropin, will be necessary. Foreign bodies can often be detected by the use of focal illumination, but may be hidden by the opacity following their entrance. Ruptures can often be seen by focal illumination. In using the ophthalmoscopic mirror at close range for the minute study of the lens it will be necessary to place a convex glass of 10 to 20 D. strength behind the mirror. If part of the iris is congenitally absent or if a peripheral iridectomy has been made, it is possible to see the ciliary processes, the suspensory ligament, and the edge of the lens.

The Vitreous Humor.—The lens being clear, the anterior part of the vitreous can be examined by the oblique method. Practically the ophthalmoscope is necessary in the examination of this humor.

With a dilated pupil gross changes can be seen by using a concave retinoscopy mirror of twenty-five centimetres' focus. If the vitreous is opaque in spots, it will be necessary to determine whether the opacities

Fig. 84.—Opacities in the lens.

A, Opacities seen by reflected light. *B*, The same by transmitted light.

are floating or fixed. The patient is told to move the eye quickly up, then down, and he is then to stop suddenly at the middle line. This will cause floating bodies to move. This maneuver shows large opacities plainly, but small, dust-like spots can best be seen by the direct method of ophthalmoscopy. Fixed opacities must be studied by the same method. The localization of foreign bodies in the eye by the use of the x-rays has come to be of great value.

The Ciliary Body.—The condition of this part of the eye is discovered by inspection and palpation. Tumors growing here often produce bulging of the corneoscleral region. If inflammation of the ciliary body is present, pressure through the intervening lid will cause tenderness and pain.

The Tension of the Eye is determined in the following manner: The patient is told to close the eyes and look downward. The examiner, sitting in front of the patient, places his index finger on the upper lid and presses on the eye at the anterior scleral region. The pressure is applied by one index finger while the other rests steadily on the lid. If the eye can be dimpled by moderate pressure, the tension is said to be normal (Tn).

Abnormally, tension may be either increased (plus tension) or decreased (minus tension). In recording the findings the following divisions and abbreviations are used :—

Tn = normal tension; T + ? = probable increase; T + 1 = tension perceptibly increased; T + 2 = tension markedly increased; and T + 3 = eye of stony hardness.

The degrees of minus tension are correspondingly recorded in this way :—

T — ?, T — 1, T — 2, and T — 3.

Tonometers—instruments constructed on the principle of the manometer—are used in physiologic laboratories for recording ocular tension, but are of little value clinically.

The educated touch *(tactus eruditus)* is necessary in correctly estimating tension. The student should avail himself of all opportunities for proficiency in this line.

Fig. 85.—Method of taking the tension of the eye.

Physiologists have found that normal intra-ocular tension equals from 26 to 28 millimetres of mercury.

Tension, as a rule, is increased in glaucoma, in intra-ocular tumors, and occasionally in iritis. It is decreased, as a rule, in detachment of the retina, in perforations of the globe, in liquefaction of the vitreous humor, and in pseudo-neuroepithelioma.

The Orbit.—The position of the eyeball should claim attention. If too prominent, it may be luxated or simply too protuberant. ***Luxation*** is present when the exophthalmos is so great that the globe has left the orbit entirely and lies in front of the septum orbitale. It is caused by trauma. A less degree of protrusion is called *exophthalmos,* and is found in orbital tumors and in suppuration in sinuses adjacent to the orbit. It is present in Graves's disease, in paralysis of several orbital muscles, in inflammation of Tenon's capsule, and in general inflammation of the orbital tissues. It may be caused by the presence of foreign bodies in the orbit. Exophthalmos can be measured by comparing the diseased with the healthy side.

In exophthalmos due to the growth of a tumor in the orbit the finger may be used as a probe and often the neoplasm can be felt. In case an aneurism is present the finger may feel the pulsation and the phonendoscope will elicit the bruit, not only at the base of the orbit, but also over a considerable area of the skull. The accessory sinuses, frontal and ethmoidal, the nasal cavity, antrum of Highmore, roof of the mouth, and upper pharynx should be examined in any case of exophthalmos. In doubtful cases of tumor of the orbit, or of the adjacent parts, electric transillumination should be employed.

The presence of foreign bodies buried in the orbit can be determined frequently by the use of the x-rays.

Enophthalmos, a sinking of the eye, results generally from trauma, but may be an idiopathic affection proceeding from lesion of the cervical portion of the sympathetic nerve. Atrophy of the orbital cellular tissue with loss of fat, loss of water due to cholera (von Graefe), and abscess of the orbit are causes. The extent of enophthalmos may be judged by comparing the diseased with the sound side.

Balance of the Ocular Muscles.—The various extra-ocular muscles are delicately balanced under normal conditions. The tendency of the recti to draw the eyeball backward is opposed by the action of the obliqui. At rest, the anatomic position of the eyes is naturally divergent, owing to the direction of the orbits and the optic nerve and the natural length of the muscles. The deviation of sleep, however, is immediately succeeded on waking by the functional position of rest, in which the visual axes become parallel. To maintain this position is easy and natural for the individual when the muscles are properly balanced. If one muscle or set of muscles is weak, an increased innervation is required to maintain the normal balance. There are many tests of the muscle-balance, but only those most generally used will be described. First, however, it is necessary to mention definitely the terms which have been applied to the anomalies of the muscle-balance. These names were proposed by Stevens:—

Orthophoria, normal adjustment of the ocular muscles.

Heterophoria, abnormal adjustment of the ocular muscles.

Hyperphoria, a tendency of one eye to rise above its fellow.

Hypophoria, a tendency of one eye to fall below its fellow.

Exophoria, a tendency of the visual axes outward.

Esophoria, a tendency of the visual axes inward.

Hyperexophoria, a tendency of the visual axis of one eye to deviate upward and outward.

Hypoexophoria, a tendency of the visual axis of one eye to deviate downward and outward.

Hyperesophoria, a tendency of the visual axis of one eye to deviate upward and inward.

Hypoesophoria, a tendency of the visual axis of one eye to deviate downward and inward.

Cyclophoria (Savage) is a want of equilibrium of the oblique muscles.

The tests for muscle-balance are to be carried on at 20 feet and 13 inches, respectively. The test objects are a lighted candle for distance, and a white dot on a black field for near. The surgeon should employ (1) the cover test, (2) the fixation test, (3) the Maddox rod, (4) the von Graefe test, and (5) the Maddox double prism. The cobalt-blue glass test is used by some surgeons. These tests are to be used while the patient possesses his full accommodative power: *i.e.,* before a mydriatic or cycloplegic is employed.

The Cover Test is applied by having the patient look at a distant object placed in the median line on a level with the eyes. An obturator is placed alternately over the eyes, and the surgeon observes the position and movement of the eye at the instant of uncovering. A movement inward indicates that the eye has deviated outward (exophoria). A movement downward means hyperphoria. The test is repeated at thirteen inches.

The Fixation Test.—The patient is told to observe the surgeon's finger placed at 13 inches from and on a level with the eyes. The finger

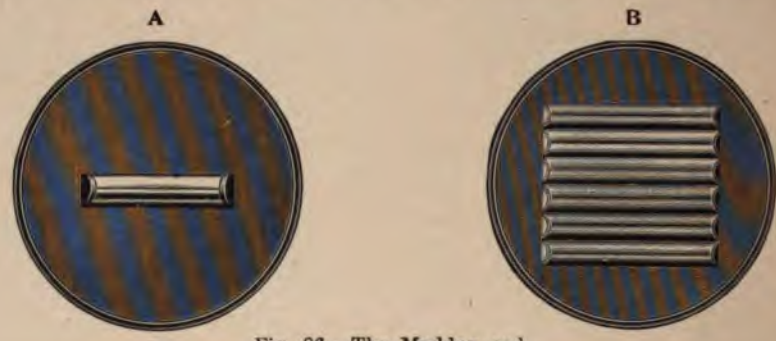


Fig. 86.—The Maddox rod.
(Courtesy of D. V. Brows.)
A, The single rod. *B*, Multiple rod.

is then advanced slowly toward the patient's nose to within $3\,^1/_2$ inches (8 centimetres). If one eye turns outward, there is exophoria. The test is a rough one, and its chief value is in determining which of the interni is the weaker.

The Maddox Rod is practically a cylinder. It causes a luminous point to appear elongated, as a line or streak. When placed vertically before the right eye, the other being uncovered, the image of the light seen by the right eye becomes a streak. The Maddox rod test is used in the examination for both vertical and horizontal deviations. It is used at 20 feet, in a dark-room, and forms a reliable, rapid, and inexpensive test. The instrument is on the market in two forms: (1) the single rod, and (2) the compound rod. The latter consists of a series of rods joined laterally. The former must be carefully centred to be in line with the visual axis; with the compound rod little care is necessary in the adjustment. In normal conditions the streak made by the rod passes directly through the

luminous point; if it deviates to the right or left, or is placed above or below the light, heterophoria exists; and the prism which corrects the faulty position of the streak is the measure of the heterophoria. This test shows esophoria to be a more common condition than exophoria.

Testing for horizontal deviations is carried out as follows: The patient is placed 20 feet from and on a level with an electric light or gas-jet covered with a ground-glass globe. The room is darkened. Let it be supposed that

Fig. 87.—Diagram to show the Maddox rod test for horizontal deviations.

The rod is in front of the right eye. *1*, Orthophoria. *2*, Esophoria. *3*, Exophoria.

the Maddox rod is carefully adjusted over the right eye, the rod being placed horizontally in a trial-frame, and the left eye being free. The patient looks at the flame and informs the surgeon of the location of the vertical streak of light. If it passes directly through the light, orthophoria exists; if the streak is to the right there is latent convergence (esophoria); if it is to the left, there is latent divergence (exophoria).

In testing for vertical deviations the rod is placed vertically before one

Fig. 88.—Diagram to show the Maddox rod test for vertical deviations.

The rod is in front of the right eye. *1*, Orthophoria. *2*, Right hyperphoria (the upper image belongs to the left eye). *3*, Left hyperphoria (the upper image belongs to the right eye).

eye. In orthophoria the streak will run horizontally through the centre of the light. In case the streak passes below the light (the rod being in front of the right eye) there is right hyperphoria; if above, left hyperphoria is present.

The von Graefe Test is made by producing vertical diplopia by placing a 7° or 8° prism vertically in front of one eye and then looking with both eyes at a dot and line on a piece of paper held at the reading dis-

tance or at an electric light at 20 feet. When using this test at 20 feet the patient in orthophoria will see two lights, one directly beneath the other. If heterophoria exists there will be a lateral displacement of the images. The prism, base in or out, which causes the images to occupy the normal position (one directly beneath the other) is the measure of the esophoria or exophoria.

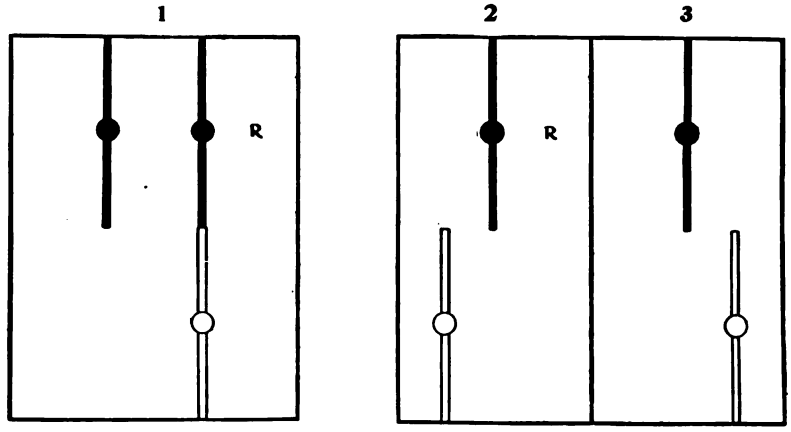


Fig. 89.—The von Graefe test, used at the reading distance.

1, Orthophoria. *2*, Exophoria. *3*, Esophoria. The prism is placed base up before the right eye. The lower image belongs to the right eye.

If used for testing muscular equilibrium for near, the patient should look at a line in the centre of which is a dot. In orthophoria the line will appear elongated and two dots will be seen. In heterophoria two lines and dots are seen, the lower set being to the right or the left.

THE MADDOX DOUBLE PRISM is used in one of the simplest tests of the ocular muscles, and is not only of value in testing the recti, but, accord-

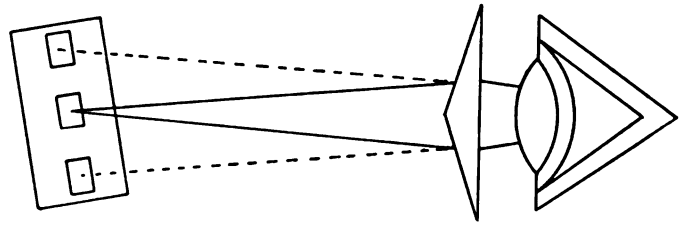


Fig. 90.—The Maddox double prism as a near test.

ing to Savage, is of use in determining insufficiency of the oblique muscles. The instrument consists of two weak prisms (3° or 6°) placed base to base in a metal rim which is of standard size to fit into a trial-frame. The double prism is to be placed horizontally before one eye in such position that the line of junction of the two prisms will be on a level with the centre of the pupil. On looking through the instrument (placed before the right

eye, for example) three images will be seen: one higher and one lower than the real image, which is seen by the left eye. To distinguish the real image it is well to place a red glass in front of the left eye. On looking at a distant object, such as a flame, the instrument being placed horizontally, vertical diplopia is produced. If the muscle-balance is normal the three images will be on a line (Fig. 91); if esophoria is present, the red light will go to the left; if exophoria exists, it will pass to the right. If hyperphoria of the left eye is present the red light will move downward. In left hypophoria the red light moves upward. In the same way the right eye can be tested by changing the position of the double prism and red glass.

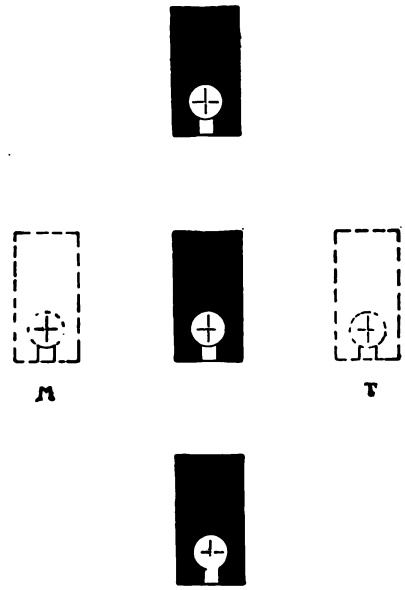


Fig. 91.—Use of the Maddox double prism for far.

The prism is in front of the right eye, a red glass being placed over the left one. The real image is the middle one, and is red. *M*, Position of the red light in left esophoria. *T*, In left exophoria.

To measure the amount of the muscular error the surgeon can place prisms of increasing strength before the eye bearing the red glass, with base in, out, up, or down, as the case may require, until orthophoria is established.

The double prism can be used as a near test for the muscle-balance, the patient looking at a line and square, or dot, placed horizontally at the reading distance. Thus, in Fig. 92, *1* represents the line and dot; *2*, the appearance of the same in orthophoria; *3*, left esophoria; *4*, left exophoria; *5*, hyperphoria; *6*, hypophoria; *7*, hypoexophoria, and *8*, hyperexophoria.

Savage has proposed to test the oblique muscles by the same means, using a Maddox double prism, each segment of which is of 6° strength.

Insufficiency of the obliques is shown by the lack of parallelism between the middle line and the others. With the instrument in front of the right eye, the other being uncovered, if the right ends of the middle and lower lines converge (*1*, Fig. 93) there is insufficiency of the superior oblique; if the right ends of the superior and middle lines converge (*2*, Fig. 93), the inferior oblique is assumed to be too weak. It is necessary to state, however, that many eminent ophthalmologists do not accept Savage's views, but regard the phenomena as physiologic.

THE PHOROMETER AND ROTARY PRISM. — These are valuable and necessary instruments for the rapid measurement of muscular insufficiency.

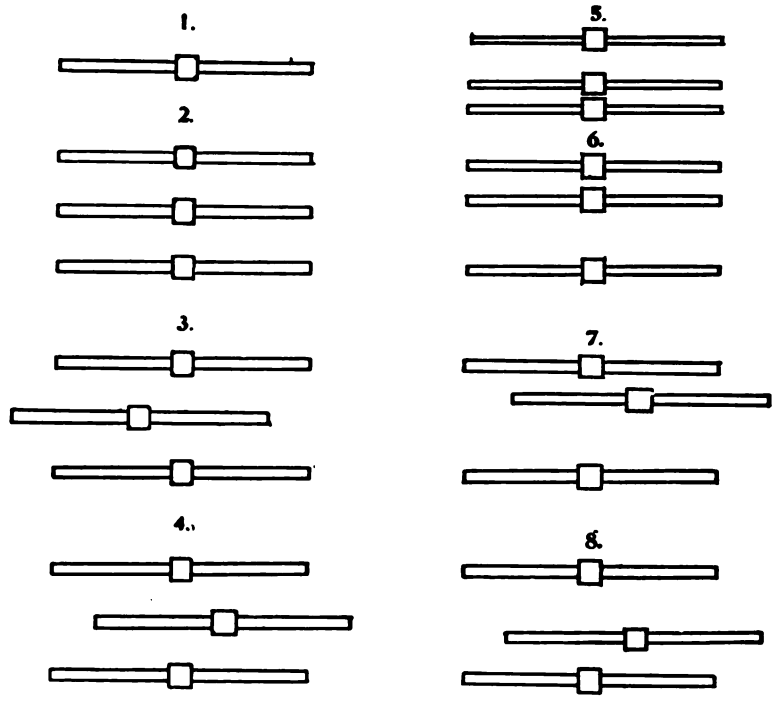


Fig. 92.—Diagram to show use of the Maddox double prism for near.

Stevens's *phorometer* (phorometer, "a measurer of tendencies") consists of two rotating discs, each carrying a prism of 5°. Each disc possesses a border of cogs, and a gear-wheel placed between the two discs causes them to move in unison. A scale, increasing from the centre each way from 0° to 8°, gives the strength of the refracting angle of the prism used. The essential part of the instrument is mounted on a leveling arm supported by a shaft and tripod. A locking device permits the arm to be lowered when the instrument is not in use. The leveling is secured by a micrometer-screw (*F*, in Fig. 95). In using the instrument the slide is placed in the groove of the phorometer, the face of the instrument being away from the patient. The side marked *RH.*, *LH.*, will then be before the patient's right eye;

while the other side, marked *ES., EX.*, will be before the left eye. After leveling the instrument, in testing for hyperphoria the pointer is placed at 0°, and the patient looks straight through the prisms at a flame placed at a distance of 20 feet. He sees two images of the flame. If these images are on a level, the muscle-balance is normal. If one image is higher than the other, the prisms are rotated until the images are made level. The

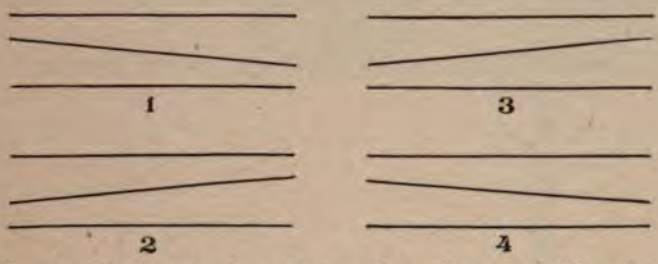


Fig. 93.—Diagram to show the use of the Maddox double prism in testing the oblique muscles.

1, Insufficiency of left superior oblique. *2*, Insufficiency of left inferior oblique. *3*, Insufficiency of right superior oblique. *4*, Insufficiency of right inferior oblique.

pointer then indicates the form and amount of the manifest hyperphoria. If the rotation is made slowly a greater amount of error will be recorded than if the prisms are moved rapidly. In examining for esophoria and exophoria the pointer is to occupy the vertical position. Normally the two images will be on the same vertical line. If one is displaced to the right

Fig. 94.—Stevens's improved rotating prism slide.

or left the adjustment is made as before until the images occupy the same vertical line, and the amount of insufficiency is read off by the scale on the side marked *ES., EX.* Other phorometers have been devised by Wilson, Lewis, and Verhoeff.

The Rotary Prism.—For rapid work in the measurement of muscular imbalance the rotary prism of Crétès or the revolving prism of Risley is

useful. These instruments consist of two superimposed prisms of the same strength, provided with a mechanism which turns them in opposite directions. When the apices coincide the strength is double that of each single prism. When the apices are opposite the prisms neutralize each other.

Fig. 95.—Stevens's phorometer ready for use.
A, Support. *F*, Micrometer-screw. *E*, Spirit-level.

Between these points any degree of deviation can be obtained, and thus the instrument replaces a whole battery of prisms. When in use it is placed in a trial-frame and the patient looks at a flame twenty feet distant, the other eye being uncovered. A scale registers the amount of error.

Fig. 96.—Risley's rotary prism.
(Courtesy of D. V. Brown.)

Power of the Ocular Muscles.—Having described the reliable tests for muscular imbalance, it is necessary to speak of adduction, abduction, and sursumduction. *Adduction* (prism-convergence) is measured by finding the strongest prism before one eye, the base of the prism being toward the temple, with which the patient can see a flame single at 20 feet. The

examination is begun with the use of a 15° or 20° prism, the strength being increased until double vision occurs. The other eye, of course, is uncovered. Normally adduction varies from 30° to 50° or 60°. *Abduction* (prism-divergence) is similarly tested, the base of the prism being toward the nose. It amounts to 6° or 8° of prism. Stevens states that the ratio between adduction and abduction should be 6 to 1, but Risley believes that in emmetropic or carefully corrected eyes it is often as 3 to 1. *Sursumduction* (sursumvergence) is the power of uniting the image of a flame seen through a prism which is placed vertically before one eye while the other is uncovered. The examination is begun with a weak prism, $^1/_2$° to 1°. It usually amounts to 3°, but may reach 10°. The amount of *right sursumduction* is equal to the strongest prism, placed base down before the right eye or base up before the left, through which the patient can see the test-object single. The amount of *left sursumduction* is ascertained similarly by placing the prism base down before the left eye or base up before the right. Right and left sursumduction should be equal.

Fig. 97.—Prism-holder. (NOYES.)

It is necessary to state that, if the patient has an error of refraction, this must be carefully corrected before the final deductions are made from the examination of the muscles. It is valuable practice, however, to examine and record the muscular condition before as well as after the examination of the refraction.

MEASUREMENT OF MUSCLE-POWER FOR NEAR POINTS.—In measuring the power of the ocular muscles for near, square prisms are used and the patient is directed to look at a point or cross placed at a distance of 13 or 14 inches. Noyes's prism-holder (Fig. 97), which is graduated into inches or centimetres and metric angles, possesses a slider carrying test-cards, and is a useful instrument. In the prism-holder before each eye are three spaces into which square prisms can be fitted. Recently Hulen has devised an improved prism-holder which permits adjustment of the interpupillary distance and can be attached to the phorometer (Fig. 98). In all cases in which the examination of the muscle-power in accommodation is made, the proper reading glasses must be used. These are called "tests in accom-

modation," and they are made on the same principle as the tests for distance. In place of the flame or electric light at 20 feet the line-and-dot test of von Graefe is used at 14 inches.

The Tropometer is an instrument for the measurement of the rotations of the eyes. Its inventor, Dr. G. T. Stevens, holds that this determination is important, since he believes that strabismus (convergent or divergent) is often due to excessive tension upon the vertically acting ocular muscles independently of abnormal tension upon the internal and external recti. He considers that many conditions of heterophoria can be similarly explained.

The tropometer "consists essentially of a telescope in which the inverted image of the examined eye is found at the eyepiece, where, either as an aërial image or as an image upon the ground glass, its movements can be

Fig. 98.—Prism-holder. (HULEN.)

accurately observed. A graduated scale in the eyepiece permits every movement of rotation, in any direction, to be exactly measured."

The scale (Fig. 100) is thus described: "The long line between and at right angles to the shorter lines divides two similarly graduated scales running in different directions; the larger circle represents the outer border of the cornea, the edges of which are in contact with the two strong lines; the interval between each pair of short lines of the scale is ten degrees of an arc, commencing at the strong line in each case. If, now, the head of the person examined is held firmly in the primary position and the eye caused to rotate strongly in a given direction, the arc through which the border of the cornea passes may be accurately read upon the scale." In Fig. 100 the curved dotted line represents a new position of the border of the cornea.

"Suppose that the person examined has been directed to look strongly

upward, then the cornea has moved down the scale and reaches the point in this example of 40°, that being the measure of this rotation. By means of the small lever the scale can be placed horizontally, vertically, or obliquely, and by means of the two graduations measurements in opposite directions can be made."

Use of the Tropometer.—In determining upward rotation the border of the cornea is made to coincide with the strong line which appears in the upper part of the scale at the right hand.

The adjustment is made by means of the milled head at the side of the standard. As the eye rotates up, the image moves apparently down. In determining the downward rotation the strong line at the lower left-hand side of the scale is taken as the point of departure.

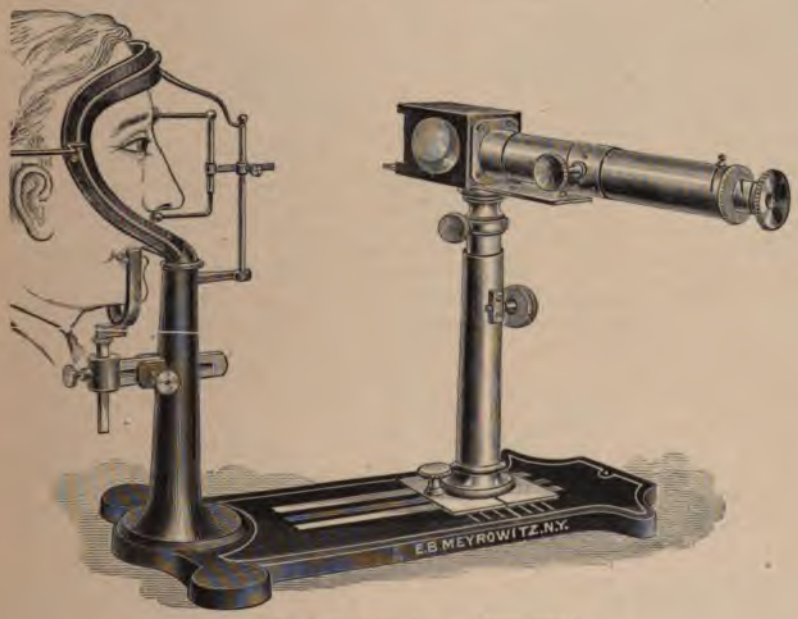


Fig. 99.—Tropometer. (STEVENS.)

For lateral rotation the scale is turned to the horizontal position and the corresponding strong lines are used as before.

In order to adjust the upper border of the cornea on the line, it will generally be necessary for the examiner to place the left hand upon the forehead of the patient and make gentle traction of the upper eyelid by the thumb. This application of the hand to the forehead is advisable in all measurements, as by this means the examiner is able to detect even a slight movement of the head, which would vitiate any measure of the rotation. In adjusting the head to the head-rest the teeth should be closed and the line of the upper lip just below the nose should be in a vertical line below the glabella, or ridge just above the root of the nose.

According to Stevens, the most favorable rotations are: upward, 33°; downward, 50°; inward, 55°; outward, 50°.

The Clinoscope.—An instrument called the clinoscope has been designed by Dr. Stevens for the testing of muscular insufficiencies. It consists of two tubes, each three centimetres in diameter and fifty centimetres in length, which are mounted on a metal platform. The tubes can be rotated horizontally and the amount of rotation is marked by a pointer and scale. The clinoscope is a valuable laboratory instrument. It is used in determining the declination of the meridians in paralysis of the ocular muscles, and in anomalous adjustments of the eyes as regards the horizontal visual plane. Stevens also uses it to determine the power of torsion and to increase torsional power by exercise.

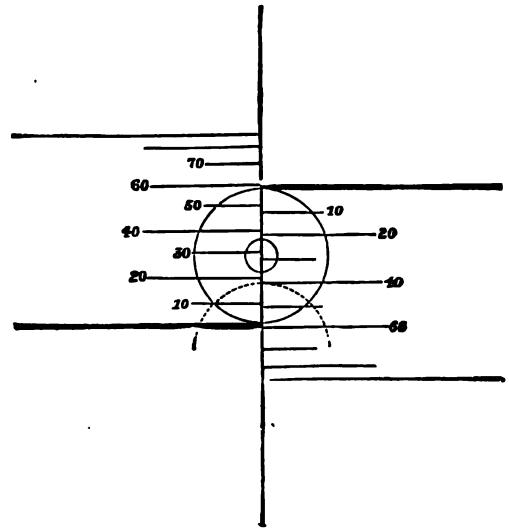


Fig. 100.—Scale of the tropometer.

Strabismometry.—Squint can be measured by any one of the following methods: (1) by the perimeter; (2) the cover, or screen, test; (3) Priestley Smith's tape-measure test; (4) Hirschberg's test; (5) the linear measurement test; and (6) the prism test. Other valuable tests have been devised by Swanzy and Maddox.

1. Measurement by the Perimeter.—In using this method, which, unfortunately, is not admissible in very young children, the requisites are a perimeter and a lighted candle. The arc of the perimeter is placed transversely across the visual axis of the deviating eye; both eyes are kept open and the normal eye fixes the middle line of the instrument. The surgeon now moves the lighted candle along the arm of the perimeter until the image of the flame is opposite the centre of the pupil of the deviating eye. The degree on the perimeter is then read off, thus giving the angle of the strabismus. While this will answer for practical purposes, to learn

the total angle of the squint requires that the angle gamma be measured and added to the angle of the strabismus. The angle gamma is the angle formed by the line of fixation with the optic axis. A perimeter and lighted candle being provided, the patient is seated as above, and fixes with the deviating eye, the other being covered. Following is Lang's description of the procedure: "The flame is moved along the arc in the same way as when ascertaining the angle of the squint. If the candle is behind the fixation-point when the image of the flame falls in the centre of the pupil, then there is no angle gamma; but if the candle is to one side of the point of fixation, when the image is opposite the centre of the pupil, then there is an angle gamma. The degree on the arc against which the candle now rests indicates the amount of the angle; if the candle is on the temporal side of the eye, the angle is positive, and if it is on the nasal side, the angle is

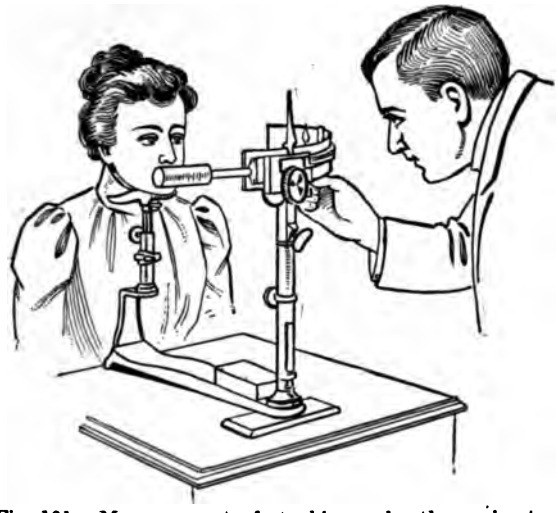


Fig. 101.—Measurement of strabismus by the perimeter.

negative. When adding the amount of the angle gamma to the angle of the squint to ascertain the total angle of deviation the positive and negative signs of the angle gamma must be treated mathematically; therefore the former increases and the latter diminishes the angle of deviation."

2. THE COVER, OR SCREEN, TEST.—The patient looks at a small distant object; the "good" eye is covered with a card, while the squinting eye fixes the object; a strabismometer is now placed on the lower eyelid with the zero mark coincident with the centre of the pupil of the uncovered eye. The "good" eye is now uncovered and fixes the object while the squinting eye turns, the amount of deviation being noted. This test is applicable to all ages.

3. PRIESTLEY SMITH'S TAPE-MEASURE TEST.—The requisites are a dark-room, a lamp, an ophthalmoscopic or retinoscopic mirror, and a double tape two metres long. One-half of the tape is black, the other half colored.

The colored half is divided into 12 parts, numbered in multiples of 5, from 5 to 60, and attached to its end is a small weight to keep it taut while in use. At the end of the black tape is a ring and another is placed at the junction of the black and colored portions of the tape. The following is the method of measurement (A. E. Davis) :—

"The patient holds the end of the black tape against his face directly under the non-squinting eye, while the observer stands directly in front of him with the ring attached at the other end of the tape over his thumb or ophthalmoscope, which he holds in front of his eye. The patient looks directly into the ophthalmoscope, from which a light is reflected into the

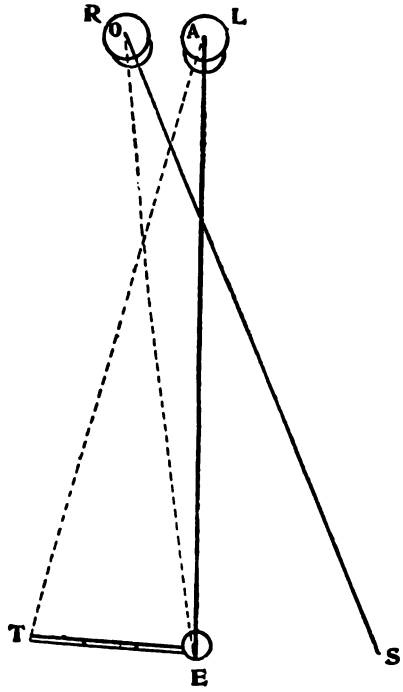


Fig. 102.—Measurement of convergent strabismus of right eye by Priestley Smith's method.

squinting eye. It will be noted that the image of the light from the ophthalmoscope will be to the outer side of the centre of the cornea in convergent squint and to the inside of the centre of the cornea in divergent squint. The observer now takes hold of the colored tape at the ring, the edge of the hand being held toward the patient for the patient to look at, and lets the tape slide between his fingers, carrying it in a direction opposite to that in which the eye squints. Both eyes should follow the hand, and, when the squinting eye has turned sufficiently for the image of the light from the ophthalmoscope to occupy the centre of the cornea in that eye, stop the hand and note the distance it has moved along the tape. The

number on the tape indicates the degree, or angle, of the squint. This method has the great advantage that it can be used on very young children."

Fig. 102 may assist in the understanding of the method. Let *R* and *L* represent, respectively, the right and left eye. The right presents convergent strabismus. The surgeon places the tape *(A-E)* in position before non-deviating eye, then throws light on to *R*, and sees the reflex eccentrically outward, which shows that this eye deviates inward. Then holding the graduated part of the tape, he moves it outward (at the same time moving the mirror) until the reflex is in the middle of the pupil. The axis of the deviating eye, *R*, will have moved from *S* to *E*, through the angle *S-o-E*. The axis of the non-deviating eye, *L*, will have moved through an equal angle, *E-A-T*. The angular movement of *L*, as measured by the tape-line, equals the angular deviation of *R*.

4. Hirschberg's Test.—This test estimates the amount of strabismus from the position of a candle-flame reflected from the cornea. The surgeon holds the lighted candle one foot in front of the patient, who has both eyes open. While the image of the flame is in the centre of the cornea of the fixing eye, it is eccentric to the pupil of the other eye. If the pupil measures 3.5 millimetres, and the image of the flame is midway between the centre of the pupil and the pupillary margin, the squint is less than 10°; if it is seen at the pupillary margin, the amount of squint is 12° to 15°; if it is midway between the pupillary margin and corneal limbus a squint of 25° is present; if the flame shows on the margin of the cornea the amount is 45° to 50°; and if on the sclera, 60° to 80°. This test is sufficiently accurate for practical purposes, and can be used on any case.

5. Linear Measurement Test. — This requires a strabismometer (Fig. 103), marked in millimetres. The patient looks at a distant object while the surgeon makes on the lower lid a vertical mark corresponding to the outer margin of the cornea of the squinting eye. Then the normal eye is covered and the patient looks at the same object while the surgeon again marks the outer margin of the cornea. The distance between these marks (distance from *S* to *R* in Fig. 104) is measured in millimetres and recorded.

6. The Prism Test can be used only when binocular vision is present (*i.e.*, in early cases and in patients in whom binocular vision has been restored by treatment). The requisites are a frame, a set of prisms, a colored glass, and a lighted candle placed at 20 feet. The colored glass is placed in front of the better eye and the patient looks at the flame. If diplopia (double vision) is present, note is made whether it is homonymous or crossed; and the prism, which produces single binocular vision, divided by two, is the measure of the strabismus. Thus, in homonymous diplopia which requires a 30° prism, apex inward, to produce single binocular vision, there is a convergent squint of 15°.

Keratometry (Ophthalmometry; Astigmometry).—By these terms is meant the act of measuring the curvature of the cornea and the determination of the difference of curvature in different meridians: *i.e.*, the meas-

uring of corneal astigmatism. Instruments for this purpose (keratometers, ophthalmometers, astigmometers) consist of a telescope, carrying a metal arc movable around its axis; a head-rest, for the support of the patient; a leveling screw, by which the telescope can be raised or lowered; a Wollaston prism, placed between two achromatic objectives, which serves to double objects in a direction parallel with the plane of the metal arc; a Ramsden eyepiece with a spider's thread; two white objects (mires), each of which is carried on an arm of the arc; and another arc which registers the position of the meridians of greatest and least refraction. These parts are diagrammatically shown in Fig. 105. In using the astigmometer a reliable source of illumination, such as gas or electric light, is required, and is generally best furnished by four Welsbach burners or the same number of incandescent lights. The author prefers gaslight, for the reason that the

Fig. 103.—Strabismometer.

Fig. 104.—Linear measurement of strabismus.

intensity of the light can be controlled. The principle on which the instrument is based, that of doubling, has been borrowed from astronomy and can only be referred to here. Originally the invention of Helmholtz, who used it solely as a laboratory instrument, the astigmometer has been made a clinical necessity by the labors of Javal and Schioetz. One of the mires is a parallelogram; the other has the special form of "insteps," each of which corresponds to one dioptre.

In making the examination only the central portion of the cornea is inspected—the part forming an area extending about 1.2 millimetres in every direction from the visual line. Hence it is necessary that the patient should look directly ahead into the barrel of the instrument and hold the eye in a fixed position. The telescope is then focused by a to-and-fro movement. On looking through the astigmometer the surgeon will see four images of the two mires: the outside images are to be ignored,

the central images are to be studied. One of the mires is fixed, the other is movable on the arc. The mire at the right is to be moved until the central images touch at their bases (Fig. 106). Then the instrument is to be rotated 90° on its axis and the relations of the images noted. If they overlap, astigmatism is present and each step thus covered represents 1 D. of error. If the images separate, astigmatism "against the rule" is present, and can be measured by causing the images to touch at their bases and then turning the instrument back 90°. If the images retain their relation, the bases touching, there is no corneal astigmatism.

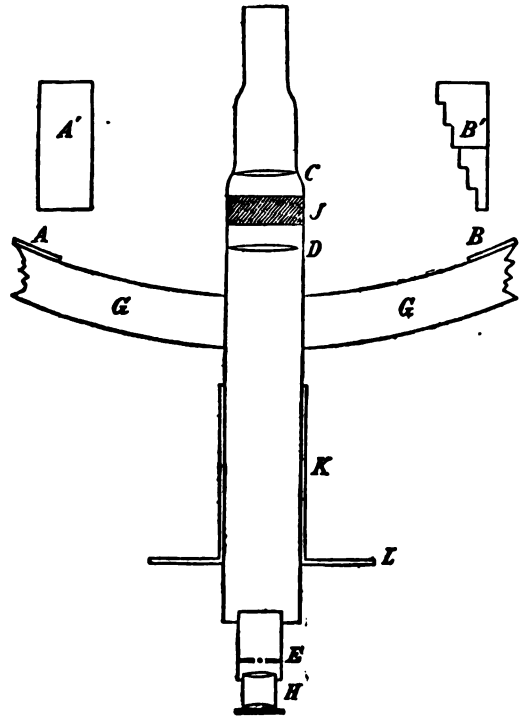


Fig. 105.—Schematic section of the Kagenaar model of the Javal-Schioetz ophthalmometer. (KAGENAAR.)

A, *B*, Transverse section of the mires. *A'*, *B'*, The same seen on the flat. *C*, *D*, Objectives. *E*, Spider's thread. *G*, *G*, Metal arc. *H*, Ocular. *J*, The doubly refracting prism. *K*, Metal tube. *L*, Arc divided into degrees.

While inspecting the cornea by this instrument it will often be noticed that the images move slightly, sometimes overlapping, then separating, showing that the curvature of the cornea can be changed by the tension of the recti muscles. This circumstance explains why the ophthalmometer often registers 0.50 or 1.00 D. more astigmatism than is actually present as demonstrated by retinoscopy and trial-lenses. In the case of a quiet patient, and in the hands of a competent observer, the discrepancy between

the amount of astigmatism found by the instrument and the glass accepted by the patient will generally be surprisingly small.

The astigmometer accomplishes several things: 1. It registers the amount of astigmatism and gives the axes of the principal meridians. 2. It unerringly detects corneal nebulæ or opacities causing irregular astigmatism. 3. It gives a magnified view of the iris. It does not tell whether the astigmatism is myopic or hypermetropic. For the examination of the deaf and dumb, children, and illiterates it is an absolute necessity, and for the rapid and correct determination of refraction in all other persons

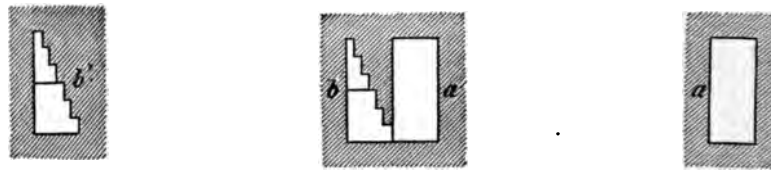


Fig. 106.—Appearance of the mires in the primary position. (KAGENAAR.)

it is useful. Although many ophthalmometers are on the market, the author prefers the one here described. The recent model of the Javal-Schioetz instrument, provided with a large disc on which confusing circles and figures are found, is not so valuable as the model of 1889.

Testing Visual Acuity.—The sense of sight consists of (1) the form-sense (visual acuity), (2) the light-sense, and (3) the color-sense.

TESTING THE FORM-SENSE.—By the term "form-sense" is meant the power which the eye possesses of distinguishing form, after the refraction has been corrected, if it be abnormal.

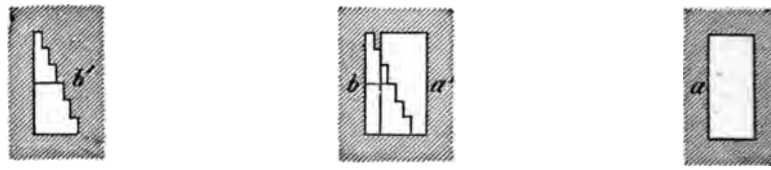
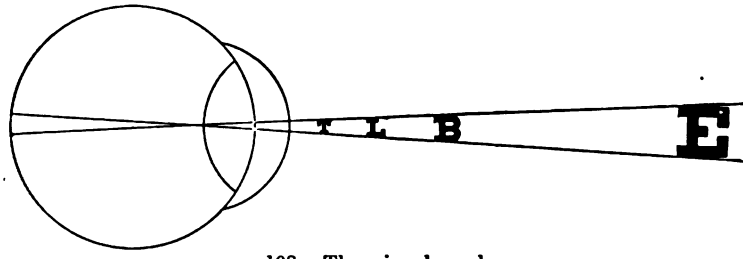


Fig. 107.—Appearance of the mires in the secondary position, showing four dioptres of astigmatism. (KAGENAAR.)

Having finished the external examination, it will next be in order to test the visual acuity of the patient. In this form of examination everything depends on the answers of the patient. Hence in malingerers, illiterates, and children this test is of doubtful value.

In order to make the examination of visual acuity of value, a definite standard is necessary. This was supplied many years ago by Snellen, whose test-types are in general use. They are so constructed that each letter is formed within a square, each side of which is divided into five equal parts. The sizes of the letters are such that the normal eye will see them at certain

definite distances under a visual angle of five minutes. Each of the five small squares is seen under a visual angle of one minute, which is supposed to be the minimum visual angle for the normal human eye. In order to subtend the same visual angle it is necessary that the letters used as tests



108.—The visual angle.

should increase in size in proportion as the distance from the eye increases (Fig. 108).

The Snellen test-types are shown in Fig. 109. Although they have been generally accepted as the standard, it has been found that letters constructed under the angle of five minutes do not always give the best visual

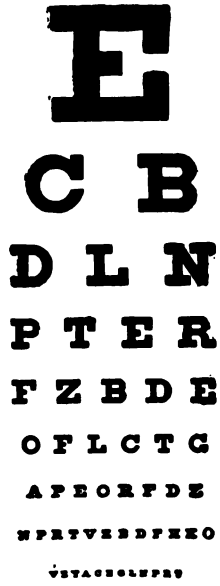


Fig. 109.—Snellen's test-type.

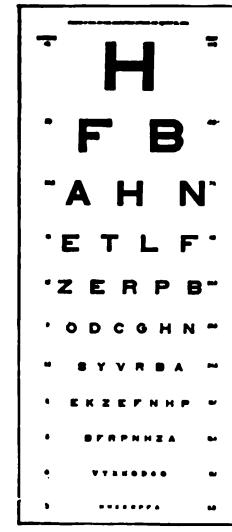


Fig. 110.—Wallace's test-type.

acuity of which the patient is capable, many eyes possessing vision $^{5}/_{4}$ of this standard. Hence, test-types have been constructed on the basis of an angle of four minutes.

The test-type of Wallace is constructed on the four-minute basis. Some objection has been raised to the intervals between the lines in Snellen's

types. This has caused Monoyer to construct a series of lines based on the decimal system in which the interval between each line is $^1/_{10}$ and the degrees run from 0.1 to 1.0. These types were highly commended by Noyes.

The letters used as tests are printed in black on a heavy white paper or painted on porcelain, or, as is now preferred by many surgeons, the letters are white with a black background. It is claimed for the latter that a white letter on the black card is not so tiring to the eye as the ordinary test-type.

For recording visual acuity Snellen furnished the formula: $V = \frac{d}{D}$. In this *V* stands for vision, *d* indicates the distance of the patient from the type, and *D* the distance at which it should be read.

Acuteness of vision is found by determining the smallest type which can be read at 6 metres. Rays of light coming from an object at this dis-

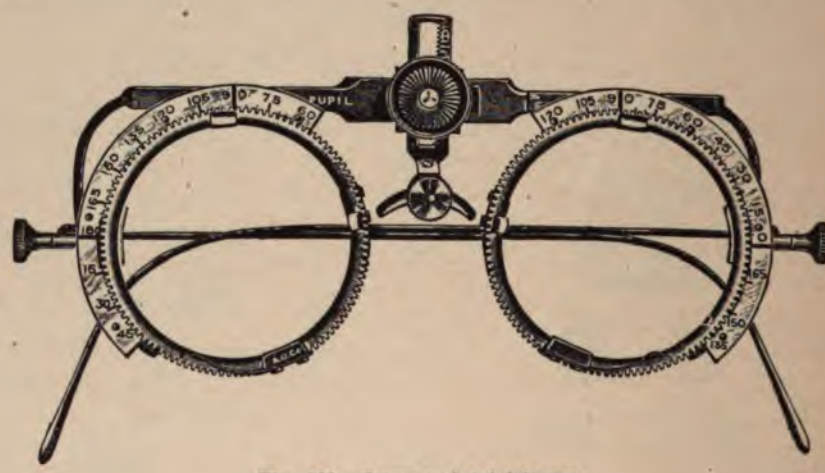


Fig. 111.—Improved trial-frame.

tance are assumed to be parallel. In the practical use of test-types it is necessary to have a steady and equal illumination. Since daylight does not answer these requirements, it is best to use artificial light. A reflector is used to throw the light from a gas-bracket on to the type. The patient, being placed at the proper distance, is requested to read the letters, beginning with the largest. The eye not under examination is covered with a metal disc placed in a frame for holding lenses, commonly known as a "trial-frame" (Fig. 111).

If the patient correctly reads the line which the normal eye reads at 6 metres, vision should be recorded as follows: RE = $^6/_6$. The left eye is then tested in the same manner. If, for example, this eye reads at 6 metres only the line which should normally be read at twice this distance, vision should be recorded as LE = $^6/_{12}$. Attempts are then to be made to render the vision normal by placing a spheric or cylindric lens, or a

combination of the two, in front of the eye. The result is recorded as part of the case history. Regardless of whether normal vision is attained by lenses or without them, it is necessary in many cases, particularly in persons under forty years of age, that a mydriatic be used in order to examine the refraction properly. Before proceeding to its use, however, it will be advisable to examine the near vision, the light-sense, the color-sense, the field of vision, the muscle-balance, and the intra-ocular tension.

In case the patient cannot read the largest letters on the test-type at 6 metres, he is to be led toward the letters, and, when the top letter is recognized, a note is made of the distance. If, on closely approaching the test-type, it is found he cannot read any letters, the surgeon is to determine whether the eye under examination can see fingers at a few inches. Should there exist inability to recognize fingers, it will be necessary to find whether light-perception, PL, exists. This can be done by taking the patient into a dark-room and throwing a faint light upon the eye by means of an ophthalmoscope or retinoscope. The mirror can be turned so as to illuminate different parts of the retina, and the intensity of the light can be varied. The result of these observations is to be recorded for future comparison. Light-perception may be qualitative or quantitative. If qualitative, the patient will distinguish between two sheets of paper, one of which is entirely white, the other with printing on it. If quantitative, he will recognize the difference between a dark and a lighted room.

Up to this point it has been supposed that the patient is an adult of average intelligence. If he be illiterate, the surgeon must make use of specially constructed test-types. A common one is that designed by Snellen, in which the letter *E* is placed in various positions and the patient is to place a metal *E*, which he holds in his hand, in the same positions as obtain on the test-type. Another test is that of Burchardt who arranged a series of dots of different sizes, in the form of groups. The latter is a more exacting test. Still another way is to have pictures of animals or common objects printed according to the visual angle. Such test-types have been designed by Ewing and Wolffberg. While they are not scientifically exact, these test-types are of practical value. As regards the examination of malingerers, the subject is of such importance as to demand consideration elsewhere in this chapter. It may be remarked here that in the examination of the refraction of illiterates, malingerers, and children, credence is to be given to the findings obtained by ophthalmoscopy, ophthalmometry, and retinoscopy.

Examination of Near Vision.—This includes the testing of the ability of the patient to read print: *i.e.*, the condition of the accommodation. The test-types for near vision are those of von Jaeger and Snellen. The No. 1 of the former corresponds to Snellen's 0.50. A very valuable card for the near test has been designed by Oliver, and consists of five divisions (Plate VI). Each division is composed of several columns, each made up of three or four words. This author describes his test as follows:

"Each word is composed of three or four letters constructed in strict conformity with the Snellen basis of letter-formation; each column of words has a purposive succession of test-letters, so arranged as to be of value in the recognition of astigmatism: each grouping of letters is composed of series of words which bear no relation to one another; and each test-letter is surrounded by a space which is equal to, or greater than, the area that is occupied by the letter itself." The letters are much clearer than those of the ordinary test-type, having been cut in steel.

In the chapter on "Physiology of Vision" mention has been made of certain terms used in describing the accommodative power (see page 68). The near point *(P)* can be found by ascertaining the closest point at which the patient can read fine type. Another method, and a valuable one, is by means of the hair-optometer, which is in common use in England, but is little known in the United States. This (Fig. 112) is an instrument resembling a miniature harp, in which the strings are replaced by hairs. It is provided with a handle and a hook, to which a dioptre steel tape can be attached. The steel tape is marked in dioptres on one side and fractions of a metre on the other. In testing the accommodation the surgeon is to proceed as follows: A trial-frame is placed on the patient and one eye is

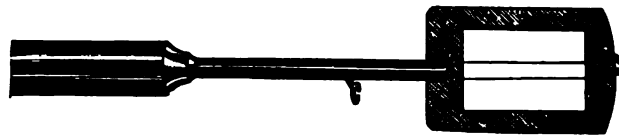


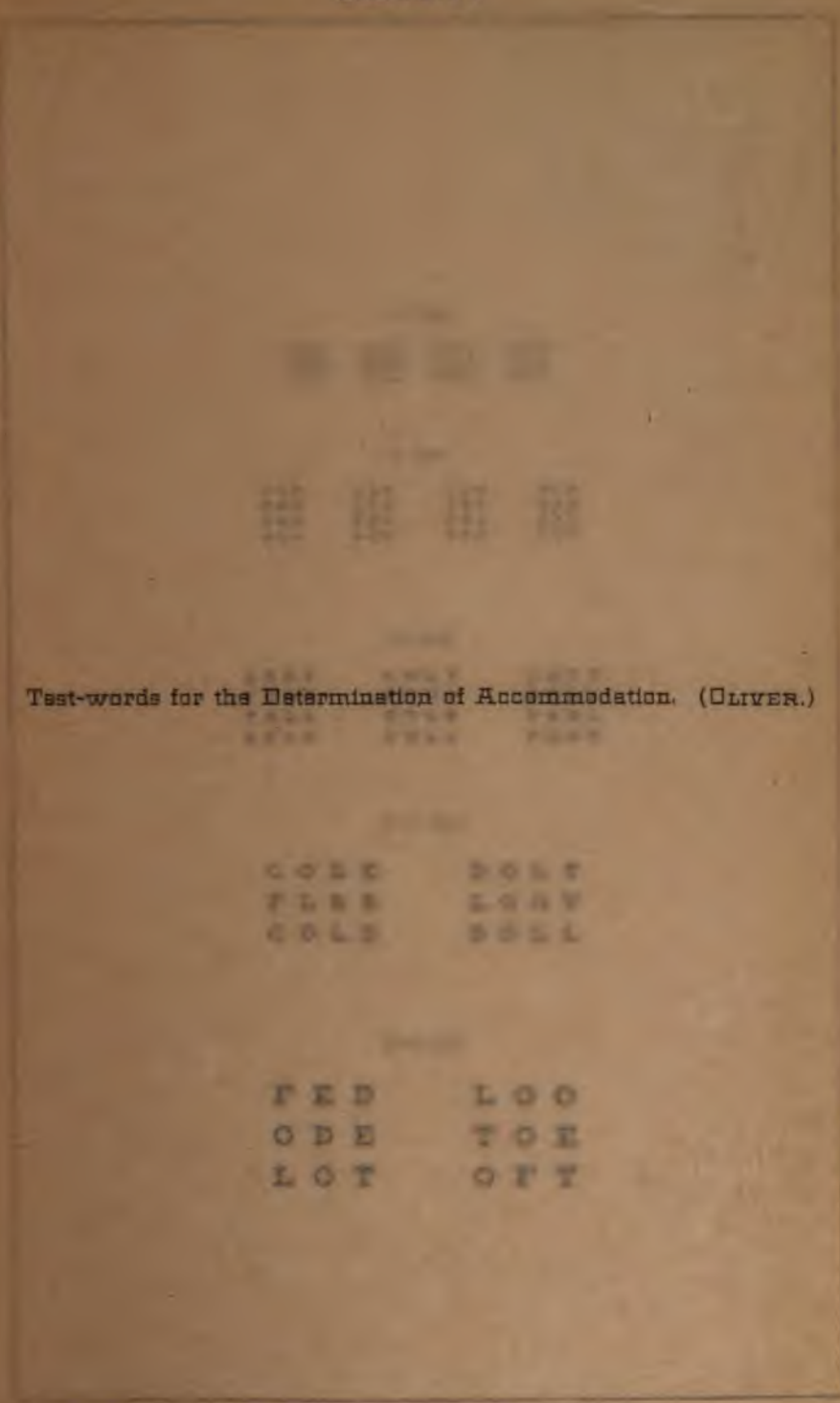
Fig. 112.—The hair-optometer.

covered with a metal disc. In front of the other eye the glass needed to make vision $^{6}/_{6}$, in the test for far, is to be placed. The patient holds the hair-optometer in front of a white background and brings the instrument to the nearest point at which the hairs can still be distinguished. The distance from this point to the outer canthus of the eye is to be read off on the steel tape. This gives the amount of accommodation in dioptres.

Suppose, for example, the nearest point at which the hairs can be clearly seen is 12 centimetres. The amount of accommodation is then $^{100}/_{12} = 8$ D. The other eye is then to be tested in a similar manner and the result is recorded. The amplitude of accommodation is determined by using the formula: $A = P - R$, in which P and R are expressed in dioptres. Suppose, in the case cited above, distant vision was made $^{6}/_{6}$ by the use of a + 1 D. spheric lens; P has already been found to be 8 D.; hence $A = 8 - 1$, or 7 D.

After the age of forty the near point recedes, and the patient is said to be presbyopic. It then becomes necessary for reading purposes to add a convex glass to the lens which is needed to correct any error in refraction. The rule is to add 1 D. for every five years after the age of forty. Thus, at forty-five the patient wears a + 2 D. lens. This rule, however, is always

PLATE VI.



Test-words for the Determination of Accommodation. (Oliver.)

Fig. 112.—The hair-optometer.

D-050

LEFT	COOT	FELT	FOOD
COOL	FEET	CLOD	LOLL
FEEL	LODE	DEFT	DOTE
DOLL	TELL	TOOL	CELT

D-075

COO	LEE	OLD	ELF
FEE	COD	LET	TOD
ODD	EEL	TOO	OFF
ELL	DOT	FOE	COT

D-100

LEET	COLT	LOFT
FOLD	FEED	DELL
TOLL	TOLD	FOOL
DOLE	FELL	FLOE

D-150

CODE	DOLT
FLED	LOOT
COLD	DOLL

D-200

FED	LOO
ODE	TOE
LOT	OFT

secondary to the following absolute injunction: Never give a patient reading glasses which magnify the print. The glasses for near work should render the object sharp and clear; they should not enlarge it. The subject of presbyopia will receive further consideration in the chapter on "Refraction."

MEASUREMENT OF CONVERGENCE.—Convergence is the power of directing the visual axes to a near point. It is brought about chiefly by the contraction of the internal recti. There is generally a close relationship between convergence and accommodation, although accommodation may be paralyzed and convergence be unaffected. Far and near points have been mentioned in connection with accommodation, and the same terms are used in describing convergence. The far point of convergence is the point at which the visual lines are directed when convergence is at a minimum, as when the eyes are directed toward an object 6 metres or farther away. The near point of convergence is the point at which the eyes are directed

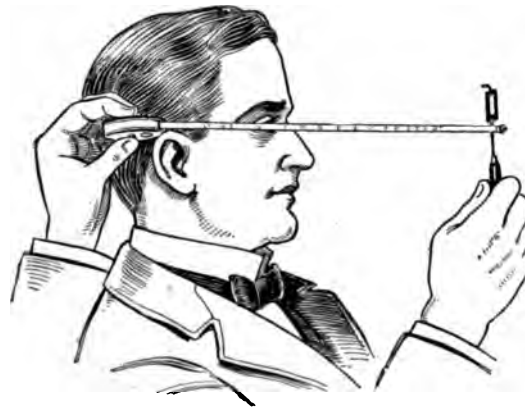


Fig. 113.—Measurement of the near point.

when turned inward to the greatest degree. It is evident that when the eyes are directed to a distant object the visual axes are parallel. If the lines diverge, they can meet only when projected backward; hence the term negative convergence. This is determined by finding the strongest abducting prism (apex of prism being toward the temple) with which the patient can overcome double vision when looking at a flame 6 metres distant. In the practical application of the test the patient keeps both eyes open, the prism being placed before one eye only. This gives in degrees the amount of negative convergence.

The term metre-angle has been used, and must now be explained. When the eyes converge to a point directly in front of them, and 1 metre distant, the amount of convergence toward the middle line is called 1 metre-angle. If they are directed to points $^1/_2$, $^1/_4$, or $^1/_8$ of a metre distant, the convergence is, respectively, 2, 4, and 8 metre-angles. Likewise, when directed to a point 2 metres distant, the convergence is recorded as

$^1/_2$ metre-angle. Accommodation for the near point is called positive convergence, and is found by having the patient hold a hair-optometer at a distance of 25 centimetres from his nose. Both eyes are to look at a bead placed on one string of the optometer; and the instrument is advanced toward the patient until the bead appears double. When this point is ascertained the distance is read off by means of the dioptre steel tape and the number of metre-angles of convergence is recorded. The average normal eye shows as many metre-angles of convergence as of dioptres of accommodation. Thus, when the eyes are directed at a point distant $^1/_4$ metre, the eye requires 4 D. of accommodation and 4 metre-angles of convergence. The relationship thus existing in normal eyes is much changed in ametropia. The hypermetropic eye calls on its accommodation before convergence is necessary. In the myopic eye the reverse is the case.

The power to maintain convergence is determined by the use of the gold bead on the optometer and a shield. The patient holds the optometer at the distance required in his work and looks at the gold bead with both eyes. The surgeon now covers one eye with the shield, which is immediately removed and the effect noted. If there is no movement of either eye convergence is maintained properly (orthophoria). If one or both eyes move when covered, and then move back to the primary position when uncovered, there is heterophoria. Its amount can be found by the following method: A word cut from Snellen's smallest test-type is pasted on a piece of white card. A vertical line is drawn through the centre of the word. A trial-frame is used; a prism of 6°, base up, is placed before the right eye. The word and line are looked at with both eyes and are seen doubled. The lower image belongs to the right eye, and should be seen immediately under the upper. If it does not occupy this position, but is placed to the right or left of the upper image, the deviation is to be corrected by placing a prism horizontally of sufficient strength to cause the images to appear in proper position.

In making a test of convergence it is necessary that a patient who wears glasses for near work shall have the lenses properly centred. If decentred, they will act as prisms and cause deviation.

Perimetry.—Direct vision having been examined by means of test-types, it will next be in order to determine the state of indirect vision. Outside the fovea visual acuity declines rapidly. Thus, according to Königshofer, 1° outside of it vision is $^1/_3$; and at 2° or 3° it is $^1/_6$. In testing vision beyond the fovea attention is paid, not to acuity, but to form. This determination is known as perimetry. While it is true that a rough estimate of the visual field can be obtained by using the hand as a test-object, and a more reliable judgment can be had by mapping the field on a blackboard (Jeffries), or with the campimeter of de Wecker, yet the best method is the use of the perimeter. This instrument is either a hollow hemisphere or a metallic band representing one meridian of such a hemisphere. The latter is a graduated arc turning on a central pivot and bearing a movable

disc. A recording apparatus registers the point at which the test-object is seen in different meridians. The perimeter of McHardy (Fig. 114), that of Priestley Smith, and that of Skeel are among the best of the self-registering instruments. Portable perimeters are of value, one of the best being Dana's.

McHardy's perimeter is used in this manner: One eye of the patient is to be bandaged. The other is to be directed constantly at a small white disc placed at the centre of the pivot. The test-object (a white disc) is then to be carried to the end of the arc and is made to approach the centre slowly. As soon as the patient sees it, the surgeon registers the point. The same procedure is followed in the upper, lower, inner, outer, and oblique meridians. To make the examination of value, at least three meridians should be tested in each quadrant. Then the points thus registered are connected by a line, and thus a map of the visual field for white is obtained.

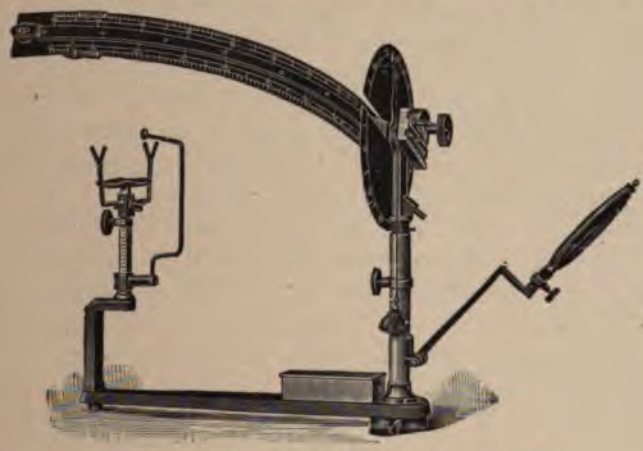


Fig. 114.—The McHardy perimeter.
(D. V. Brown, Philadelphia.)

The field for colors is tested in the same manner, a colored test-object being substituted for the white one. The apertures of the McHardy perimeter permit the use of a test-object of different sizes, 1, 2, 5, 10, 15, and 20 millimetres square.

In ordinary perimetry the test-objects are comparatively large, subtending an angle of 2° to 4°, thus covering thousands of retinal elements. Such tests are rough as compared with the tests for visual acuity. Hence a method of perimetry has been introduced by Bjerrum which involves the use of white objects subtending a very small visual angle, the examination being made at a distance of 2 metres and upon a black screen 2 metres broad. The examination is begun at the ordinary distance (30 centimetres) with the 10-millimetre white square and continued at 2 metres with the 3-millimetre disc. In the first case the visual angle is $^{10}/_{300}$ and in the second $^{3}/_{2000}$, or 2° and 5′, respectively. In the first instance the boundaries

of the normal field are those given on page 78; in the second they average 35° outward, 30° inward, 28° downward, and 35° upward (Berry). Thus, by the Bjerrum method the field is smaller; hence it is capable of giving valuable data for diagnosis.

The form of the normal field is oval and its greatest extent is on the temporal side. In case the patient is amblyopic or cataractous the examination is made by fixing one candle in the middle line while another is moved along the arc. The perimeter is useful in examining the angle of deviation in strabismus, as has been explained.

Defects in the Visual Field are of two kinds: (1) the physiologic blind spot and (2) pathologic defects. The blind spot is found 15° to the outside of and 3° below the point of fixation. Pathologic defects may take the form of concentric contraction, hemianopsia, sector-like defects, or scotomata. Hemianopsia (half-sight) can often be made out by a rough test, such as the use of the hand. The other defects require careful perimetry. While a full consideration of hemianopsia is naturally out of place in this chapter, it may be well to state briefly that the dividing-line between the blind and seeing field may be horizontal or vertical. Vertical hemianopsia may be homonymous, bitemporal, or binasal. If homonymous, both right or both lateral half-fields are absent. Absence of both right half-fields shows blindness in the left half of each retina, a condition to which the term "right homonymous hemianopsia" is applied. Left homonymous hemianopsia means blindness of the right half of each retina. In bitemporal hemianopsia both temporal fields are wanting, and in the binasal form the nasal half of each field is wanting. Further consideration of the subject will be found in the chapter on "Diseases of the Optic Nerve."

Concentric contraction and sector-like defects can be mapped out by the careful use of the perimeter. Scotomata are areas of partial or complete blindness lying within the field of vision. They are divisible into positive and negative, true and false, central and peripheral. A positive scotoma is present as a cloud which obscures vision in a certain direction and is due to retinal disease. A negative scotoma is a space in which objects naturally are unseen by the individual in health. The physiologic blind spot forms a negative scotoma. True scotomata are due to lesions in the brain, optic tracts, optic nerve, or retina, while false scotomata are caused by the obstruction arising from the presence of a blood-clot, an opacity in the dioptric media, or a new formation floating in the vitreous body. Such a defect changes its position with every movement of the eyeball. Central scotoma is due to retinal or chorioidal disease; the toxic action of alcohol, tobacco, and other substances capable of producing amblyopia; or to a form of neuritis involving the optic nerve behind the globe. Such scotomata are mapped out with difficulty because of impaired fixation.

In all cases of scotomata it is to be advised that the surgeon, after determining the extent of the field, should use a small test-object, which is passed from the centre of the perimeter outward in many meridians.

Testing the Light-sense.—The testing of the light-sense was at one time regarded as a scientific curiosity, but of late years it has come to occupy a practical place in ophthalmic examination. By the light-sense is meant the ability of the eye to distinguish different intensities of light. Two persons may have equal acuteness of vision,—*i.e.*, equal space-sense,—and yet under feeble illumination one will not discern Snellen's letters, while the other will read them. In this case the persons have a different appreciation of brightness: *i.e.*, the light-sense *(L)* is different in the two. Instruments for the purpose of comparing the intensity of one light to another, which is taken as a standard, are called photometers. In the practical application of photometry it is the sense of stimulation, not the sense of contrast, which is measured: *i.e.*, the power to distinguish the effect produced by the smallest possible quantity of light where the surroundings are dark. Since daylight is an uncertain quantity, photometers are constructed in such a way that the illumination is produced by a normal candle (one of one-candle power). The instruments of most widely accepted use are those of Förster and Henry. In the use of either it is necessary first for the patient to sit in a dark-room with bandaged eyes for ten minutes beforehand, in order that the retinæ may become adapted to darkness.

Förster's photometer (Fig. 115) is a box measuring $^1/_8$ meter by $^1/_4$ meter by $^1/_6$ meter. It is blackened inside, and provided with two apertures for the eyes to be tested. A window for the admission of light from a candle placed in a separate compartment, and black test-marks on a white ground, complete the apparatus. The test consists in finding the smallest apertures admitting the candle's rays which will permit the recognition of the test-letters. The size of the aperture is recorded on a scale marked in millimetres. Should one eye see the test with an aperture of 1 square millimetre, and a second eye see it only when the aperture is enlarged to 4 square millimetres, the second eye possesses functional power four times greater and a light-sense four times smaller than that of the first eye.

The Henry photometer consists of a box provided with an aperture, a candle, and nine discs of opal. After the preliminary bandaging of the eyes, the eye not under examination being covered, and the head enveloped in the hood, "the opal discs are, one by one, removed, and the patient is told to say when he detects any light; should he detect it through seven opals his light-perceptive power is registered as 7; if through six, five, or four, etc., 6, 5, or 4 is entered; a note is also made of the sex and age of the patient, and the condition of the fundus" (Henry).

The light-sense is diminished in many cases of general disease in which the blood is in a vitiated condition. It is often diminished in chorioidal, retinal, and optic-nerve diseases.

Testing the Color-sense.—For the investigation of the color-sense, it is useless to ask the patient to name the colors. It is only by the process of matching colors that the color-sense can be rapidly and properly tested. There are three plans for the recognition of subnormal color-perception:

(1) direct comparison of pigment-colors; (2) direct comparison of spectral colors, and (3) the study of subjective after-color (complementary color). Of these, the first, for practical work, is the most worthy of consideration. The second method is not likely to become popularized, because of the expensiveness of the apparatus, its liability to get out of order, the fact that spectral colors are not such as the patient daily observes, and the further reason that the use of the apparatus demands intelligence on the part of both the examiner and the person examined. Hence, pigment-colors must be used for comparison. The third method is of value only in exceptional expert cases.

The direct comparison of pigment-colors has been popularized by Holmgren, who borrowed the ideas of Seebeck and the worsteds of Wilson.

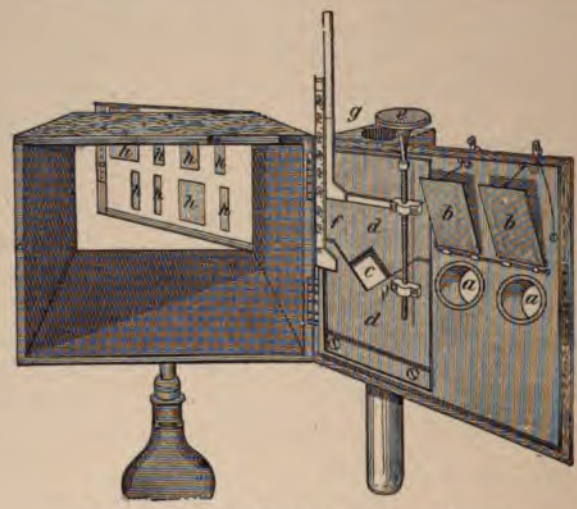


Fig. 115.—Förster's photometer. (FICK.)

The instrument is here shown with the doors open. When in use they are closed.

Holmgren's test, which has long had the prestige of authority and popularity, is not reliable. His colored plate explanatory of tests for color-blindness, which is found in many text-books of ophthalmology as a test, should not be used as such, since its colors are only intended to show the mistakes which a patient with subnormal color-perception might make. Among those who have shown the inefficiency of the Holmgren tests are Oliver, of Philadelphia; Edridge-Green, St. Clair Buxton, and G. A. Critchett, of London; and T. H. Bickerton and Karl Grossman, of Liverpool. Williams, of Boston, in one year's work, found four patients who passed the Holmgren tests, but made so many mistakes with the reds and greens of the lantern test as to show them unfit for responsible positions. The fact is that the worsteds, whether used as described by Holmgren, or as modified in Thomson's stick, or in other devices, do not represent in any

manner the actual working conditions under which color-vision is demanded. The miniature lantern tests, while more nearly attaining accuracy, are open to the same objection. In fact, the ideal way to test the color-perception of railway and steamship employees, as has been shown by Oliver, is to examine candidates under their actual working conditions. The sooner this fact is recognized, the better for the traveling public.

With reference to the determination of subnormal color-perception, the ophthalmic surgeon should divide his patients into two classes: (1) those who are to be tested as a matter of clinical routine, and (2) those who are employees of railway or steamship companies. It is evident that a method of examination which would answer for the first class would be entirely inadequate for the second. For general clinical purposes a scientifically arranged collection of loose wools is the best, cheapest, and most available material for practical use. The series devised by Oliver offers distinct advantages over many tests, in that the candidate is made to expose his color-defect by handling a series of inexpensive wools. The colors in these wools have been made of equal relative intensity; the value of each color is expressed; the set is so constructed that it can be employed by any educated layman ; notes of the passing color-changes can be preserved for future comparison, and written expressions of the character and *amount* of the perception can be given. They have all been made of one grade of manufacture, dyed with vegetable material. Properly, he employs a black surface during the testing, which is done in the ordinary manner of having the candidate select the nearest matches to the test-skeins, the designations of his choice being placed upon suitable blanks made for the purpose.

In regard to the testing of the color-sense in marine and railway employees, the candidate, as Oliver says, should be placed as nearly as possible under the actual conditions demanded by his vocation. It is evident that miniature lantern tests placed at fifteen or twenty feet, containing colored glasses of unknown ratios, while more valuable than the ordinary clinical tests, do not in any manner answer the requirements. The recognition of the colored signal must be made at a distance sufficiently great to permit the stoppage of a rapidly moving train, trolley car, or steamship. For this purpose Oliver has designed a fixed apparatus which can be used on any railway. The method is described best in his words:—

"Wooden frames containing properly and proportionately sized match and confusion colors of bunting for daylight work, or illuminated plates and lanterns of transmitted color for bad weather or night, are to be lined in a row in any order whatsoever, just as the wools are promiscuously thrown upon a table. The five test-colors similar to the ones I have employed for the near tests are placed in an upper tier. Just as with the near-work tests, the candidate employs one eye at a time. This done, he is then made successively to designate by the actual position of the color in the lower line the nearest numerical match to each of the upper test-colors. This selection

by number is to be handed to the examiner, who after having obtained the true color-names of the numbers chosen for the occasion by the attendant places the choosings upon suitable blanks for permanent registry.

"To obtain the different percentages of light-stimulus and to simulate as nearly as possible changes in character of weather (fog, rain, etc.), variously tinted glasses can be used, although preferably the candidate can by this plan be tested during the actual states of weather.

"An experimental track with a number of open switches so arranged that the sidings pass directly beneath certain colors would be useful for practically testing the color-vision that is necessary to employ while running locomotives, trolley cars, etc., at full and even high rates of speed.

"In marine service the danger is increased. All vessels carry a green light on the starboard side, and a red light on the port, each being so boxed as to be seen forward and amidships. These are accompanied by a low white

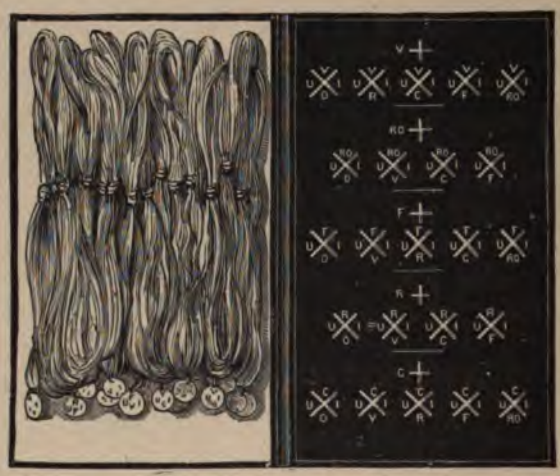


Fig. 116.—Wools for the detection of color-blindness. (OLIVER.)

forelight, and sometimes by a high white aftlight. Hence, by comparison, a vessel's course can be easily distinguished. If, under such conditions, however, a heavy fog, or snow, or a rainstorm were to exist, it can be understood how an official with subnormal color-perception, judging these important color distinctions by their intensities alone during the best of weather, is placed in a position that practically amounts to the absence of signals."

LANTERN TESTS.—Since many color-blind persons can pass the Holmgren examination when placed at 1 metre distance in good daylight; and since such persons are dangerous when occupying responsible positions in railway, marine, and naval service, attempts have been made to produce such tests as will expose deficient color-perception in this class of individuals when placed under surroundings similar to those met with in their vocations. It has been assumed that such conditions can be imitated by the

lantern tests. Lanterns for examining color-perception are used at a distance of 5 metres. By means of discs containing apertures for colored glasses over which smoked lenses can be superimposed, an attempt is made to imitate the appearance of railway signal-lights.

The lantern test will detect small defects in the color-perception of the central part of the retina which may be overlooked by the worsted tests; but, as has been said before, in the practical examination of railway employees and sailors neither the worsted nor lantern test can insure satisfactory results.

Measurement of Vision for Colors.—Apparatus for the quantitative estimation of the color-sense has been designed by Donders, de Wecker and Masselon, Truc and Valude, Oliver, and others. The examination is to be made by placing the patient at a distance of 5 metres and exposing a color through an aperture of definite size. The person is to name the color; if he cannot do so through the opening of standard size, the opening is enlarged until he is able to name the exposed color. Oliver's apparatus is

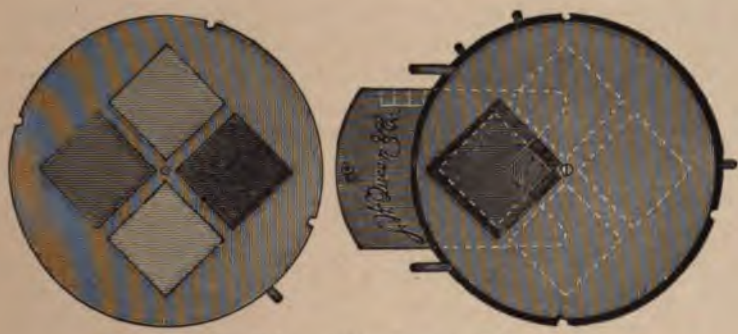


Fig. 117.—Color-sense measure. (Oliver.)

a convenient device for practical use. With it the patient, with normal color-sense placed at 5 metres, requires an opening exposing $2\,^{2}/_{3}$ millimetres of surface for the recognition of red; $8\,^{3}/_{4}$ millimetres for blue; $10\,^{3}/_{4}$ millimetres for green; and $22\,^{3}/_{4}$ millimetres for violet.

The Use of Mydriatics.—In many cases of suspected ocular disease it is impossible to make a complete examination without resorting to the use of certain medicines which act upon the iris or upon the accommodation, or upon both. Those which cause the iris to retract, thus enlarging the pupil, will be considered first; they are known as mydriatics. If they possess the property of paralyzing the ciliary muscle (accommodation), they are called cycloplegics. All of the latter class are mydriatics, but not all mydriatics are cycloplegics. Mydriatics are used for four purposes: (1) to dilate the pupil and thus permit the examination with the ophthalmoscope; (2) to paralyze the accommodation and thus permit the observer to measure the refraction by some methods which can be employed only when the ciliary muscle is made inactive; (3) to determine the diagnosis of "eyestrain"; and

(4) they are employed as therapeutic agents in inflammations, particularly those of the uveal tract.

The mydriatics employed by practical ophthalmologists are atropin, scopolamin, homatropin, duboisin, hyoscin, daturin, hyoscyamin, mydrin, and euphthalmin.

Use of Mydriatics for Refraction Purposes.—Mydriatics can be used in solid powder dropped into the conjunctiva, in oily menstruum, in gelatin discs, or, preferably, in freshly prepared solutions. In applying a solution of any mydriatic it is best to drop the medicine upon the upper part of the cornea while the patient is looking down, thus causing the solution to spread over the cornea. Applied in this way, absorption of the drug occurs more rapidly and more powerfully than when dropped into the conjunctival sac. It is always advisable that the patient should turn his head downward and to the right when the physician applies drops to the right eye, and to the left when applied to the left eye, so that the excess will flow away from the lacrimal apparatus.

An imperative rule is: *Never use a mydriatic until after testing the tension of the eye, and never use a mydriatic if the tension is increased.* Whenever possible, an ophthalmoscopic examination should be made before a mydriatic is used. No one except a skilled physician should prescribe a mydriatic, for the weakest of these drugs has been known to cause outbreaks of glaucoma. Mydriatics are dangerous remedies in the very young, in the feeble, or the aged, and should be used with discretion at all times.

Atropin, the best known of the mydriatic group, and the one whose effects last longest, is the active principle of *Atropa belladonna.* The pure atropin, owing to its slight solubility, has been replaced by the sulphate. For the purpose of examining the refraction, the use of the ophthalmoscope, etc., a solution of atropin of the strength of gr. ii-iv to the ounce is employed. A few drops of this are instilled into the conjunctiva, and a half-hour later the ophthalmoscopic examination can be made. If the refraction is to be measured, it will be best to have the patient use the solution thrice daily for three or four days. The medicine should be dropped into the outer part of the conjunctiva, or on to the outer part of the cornea, to prevent leakage into the nose and throat. Atropin solutions should be marked with a poison-label and kept under lock. The effect of atropin begins to pass off at the end of four days, and is completely recovered from at the end of two weeks. While in children and adults rapid dilation of the pupil follows the use of atropin or homatropin, in infants the application of a mydriatic is often locally unsuccessful. Although the physiologic effects may be manifested by flushing of the face, dryness of the mouth, and accelerated cardiac action, the pupils may dilate but slightly or not at all.

Scopolamin is found in belladonna-root, stramonium-seeds, and occasionally in *Duboisia myoporoides.* It is a much more powerful drug than atropin, and is used in solution in the strength of 0.1 to 0.2 of 1 per cent.

In children and feeble persons solutions of the strength mentioned may cause rapid and irregular heart-action, staggering gait, dryness of the throat, and somnolence. The effect of scopolamin begins to disappear in twelve hours and is entirely absent after five or six days.

Homatropin is a derivative of atropin, and is used in the form of the hydrobromate. It is much weaker than the mydriatics just mentioned, and its cycloplegic action is less reliable. Nevertheless, it is a valuable agent. It is used in a 2- or 2.5-per-cent. solution. To be efficient its use must be forced. A drop is placed in the eye to be refracted every five minutes for an hour; even in small doses it produces conjunctival irritation, while larger amounts will give rise to uveal and retinal disturbance (Stewart and Oliver). This is lessened and the desired action of the drug is assisted by the simultaneous use of a cocaïn solution. For making an ophthalmoscopic examination a smaller quantity is necessary. The advantage of homatropin is that its effects wear off sufficiently in twenty-four or thirty hours to permit the patient to resume his vocation. Its great disadvantage is that in many cases, used in non-irritative amounts, it does not completely paralyze the ciliary muscle. As a therapeutic measure, if employed alone, it is without value in iritis and iridochorioiditis. It may cause staggering gait, partial collapse, and hallucinations, and in some cases prolonged mydriasis.

Cocain produces a transient dilation of the pupil sufficient for the use of the ophthalmoscope, and a slight and transient paralysis of the ciliary muscle.

Duboisin in the form of the sulphate is used in the strength of gr. ss to 3ij. Its mydriatic effect diminishes at the end of forty-eight hours and is entirely absent at the end of a week. As a cycloplegic it is much stronger than atropin and its effects disappear more rapidly.

Hyoscin and Hyoscyamin are powerful mydriatics. They are said to possess marked toxic properties. Risley finds that for ophthalmic purposes only the pure crystals of hyoscyamin are to be used, and the solution should be strictly neutral, filtered through neutral paper. The solution should not be heated. An untoward effect of hyoscyamin, used in undue strength, is the production of ciliary spasm causing intense pain. The pupil is widely dilated, but the ciliary muscle is not at rest. Under such circumstances the use of atropin will relieve the pain and cause cycloplegia.

Daturin in its physiologic action is very much like atropin, and can be used interchangeably. By some it is supposed not to have any effect on the secretion of milk, and hence it is used by them in nursing women.

Mydrin is used for prompt dilation of the pupil without disturbing the ciliary muscle. It is a white, soluble powder composed of ephedrin hydrochlorid and homatropin hydrochloride: $^1/_{100}$ part of the latter to 1 part of the former. A 10-per-cent. solution promptly dilates the pupil and has little or no effect on the accommodation. At the end of thirty minutes the pupil is widely dilated, and in from four to six hours the effect has

disappeared. In young subjects there is no apparent loss of accommodation; in elderly persons there is often a transient ciliary paresis. It possesses two additional advantages: it is a time-saver and its strength is long preserved.

Euphthalmin is used in 2-, 4-, or 10-per-cent. solution for ophthalmoscopic examination. It dilates the pupil in a half-hour, and its effects pass off in ten to twelve hours. It causes only a slight disturbance of vision.

The Use of Miotics.—The agents used to contract the pupil are eserin, physostigmin, pilocarpin, and arecolin. The first three are used in strength of gr. ss to ʒvj; and the last in from $^1/_2$- to 1-per-cent. solutions. Arecolin is the best miotic for general use. Eserin may produce intoxication, tonic convulsions, hallucinations, and delirium, particularly in aged and feeble persons.

Ophthalmoscopy.—In 1851 Helmholtz predicted that by the use of the ophthalmoscope "all the pathologic changes in the retina and vitreous humor, so far observed only in the cadaver, can be seen in the living eye: a fact which promises great progress in the little-known pathology of the organ." His ophthalmoscope, which was a very simple instrument, consisting of three plates of thin glass fastened together and mounted at an angle of 56° on a brass disc, has undergone numerous modifications and improvements. These have embraced all types, such as refraction, binocular, auto- and even demonstration instruments. Demonstration ophthalmoscopes are useful laboratory instruments. Thorner's stationary ophthalmoscope surpasses other instruments of its class in the combination of a large field, brightness of illumination, and absence of reflexes. This instrument is particularly valuable in that the ophthalmoscopic picture can be made visible to any person.

Description of the Ophthalmoscope.—The ophthalmoscope practically consists of a perforated mirror attached to a handle. The mirror presents a circular perforation at its centre, and is mounted on a swivel in order that it may be turned. The mirror need not be large for direct ophthalmoscopy, since the useful part is a small area surrounding the perforation. The sight-hole should be 3 millimetres in diameter. A small opening is more useful in studying the fundus, while a larger one is preferred for the measurement of refraction. When the examiner and the patient are emmetropic, such a simple ophthalmoscope answers admirably for the examination of the fundus. In many cases, however, it is necessary to place a lens behind the mirror in order to correct the patient's refraction. It is assumed that the surgeon is emmetropic; if not, he should wear suitable glasses. For the purpose of carrying a number of lenses in a small space, rotating discs, which enable the surgeon to increase or decrease the lens-strength rapidly, have been designed. In this manner the myopia or hypermetropia of the patient can be rapidly corrected, the range of the lenses usually running from 0.25 D. to 23 D. Such instruments are called refraction ophthalmoscopes. Accompanying the instrument is a double

convex lens of from 13 D. to 20 D. strength, for use in indirect ophthalmoscopy and to obtain oblique illumination. This lens should measure $2\,^1/_2$ inches in diameter and should be supplied with a handle.

Every teacher of ophthalmology is often requested by students to specify his choice of ophthalmoscopes. It is perhaps unnecessary to say that all of the instruments on the market are useful and that good work can be done with any of them. The instrument of Loring is deservedly popular in this country and that of Morton is much used in Great Britain.

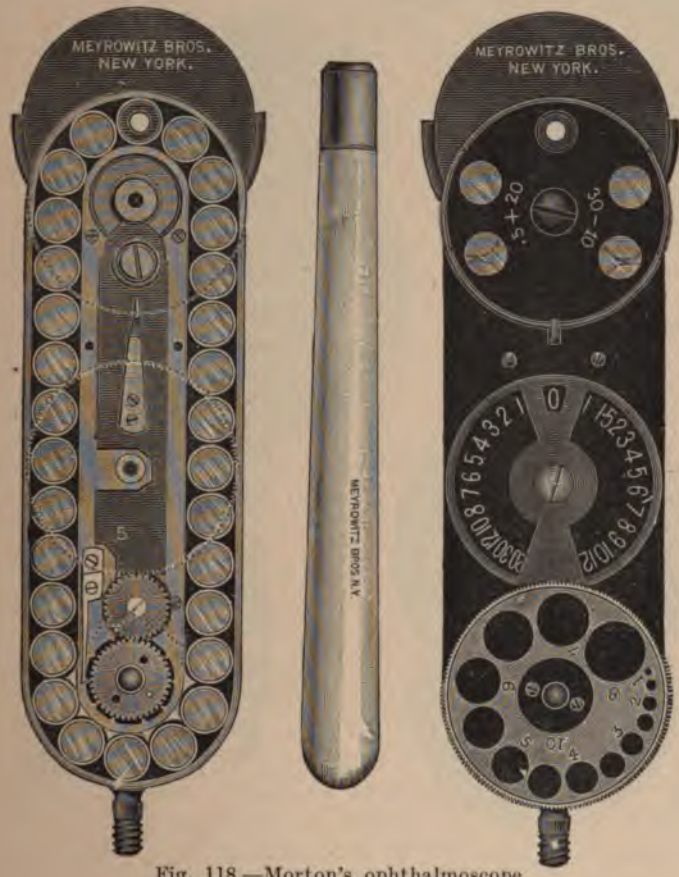


Fig. 118.—Morton's ophthalmoscope.

Excellent instruments have been devised by Couper, Harlan, Jackson, Knauer, Knapp, Landolt, May, Pyle, Randall, Snell, and others. Detailed accounts of these ophthalmoscopes can be found in the catalogues of the instrument-makers.

Methods of Use.—Not all of the light entering the pupil is absorbed by the chorioidal pigment; a certain amount returns from the eye. If the examiner's eye is placed in the same position as that occupied by the source of illumination, or immediately behind it, the interior of the eye becomes

visible. This is the principle of the ophthalmoscope. A mirror in which a hole is cut is brought in front of the eye. The mirror, gathering rays of light from a point of illumination, becomes a secondary source of light which is projected into the dilated pupil. The observer's eye, placed behind the mirror, receives these rays from the patient's eye.

There are two methods: the direct and the indirect. In the former the surgeon places his eye close to the patient's eye and looks directly upon the much enlarged and upright details of the interior of the observed eye.

Fig. 119.—Loring's ophthalmoscope.
(D. V. Brown, Philadelphia.)

In the latter he has the patient removed an arm's length, and usually a convex lens is placed between the patient's eye and the examiner's mirror. The image obtained by this method is inverted and in the air. As the direct method gives a larger magnification it shows a smaller part of the fundus at one time; it is valuable for the study of minute changes and for the practical estimation of refraction. By the indirect method the portions seen are less magnified, and hence include a larger area. This plan is the better for obtaining a general idea of the condition of the fundus with the

location of any lesions. Refraction estimated by this method is both a complicated and an uncertain procedure.

The direct method is explained in Fig. 120. Light from *L* is collected by the ophthalmoscope, *OO*, and reflected into the patient's eye. It forms an illuminated spot at *oo*. From this rays pass to the retina of the examiner

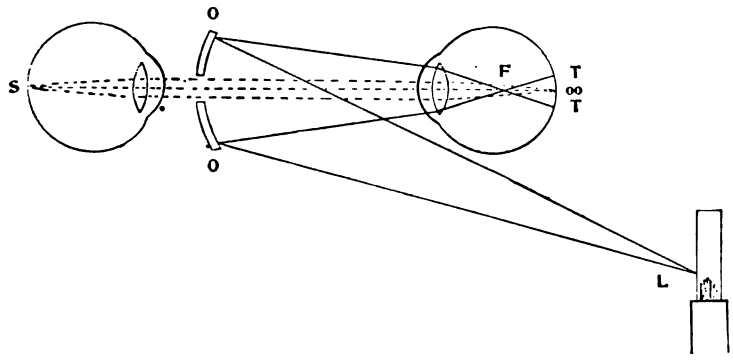


Fig. 120.—Direct ophthalmoscopy.

at *S*. These rays, coming to a focus in the vitreous humor at *F*, diverge to form an image which extends from *T* to *T*.

Fig. 121 explains the indirect method. Rays from the lamp, *L*, are reflected by the concave mirror, *Oph*, and brought to a focus at *Z*. These rays diverging from *Z* are made parallel by a convex lens *(Lens)*. The rays are brought to a focus on the retina by the dioptric arrangement of

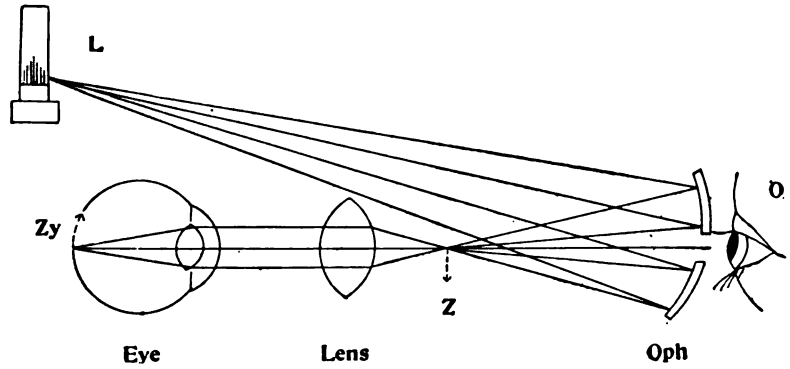


Fig. 121.—Indirect ophthalmoscopy.

the examined eye. The rays emerging from the patient's eye follow the same path and will come to a focus at *Z*. At this point *(Z)* the examiner will see an inverted aërial image.

THE ROOM AND LIGHT.—Although many do not deem it necessary, the room in which the ophthalmoscopic examination is to be made should be entirely dark. The light is preferably obtained from an Argand burner

or a frosted electric bulb, and should be steady and clear. The burner should be mounted on a bracket permitting universal movements.

Position of Examiner and Patient.—In using the direct method the surgeon should approach as closely as possible to the eye of the patient. The surgeon uses his right eye to examine the patient's right. The source of illumination must be on the same side as the eye to be examined, and on a level with it. The mirror of the ophthalmoscope should be tilted so as to face the light. The patient is told to look straight ahead, keeping both eyes open and fixing a distant object on the wall in front of him, and holding his eyes still. The surgeon should then place the ophthalmoscope in such a position that its central opening will coincide with the pupil of the eye to be examined. The proper position of the patient, surgeon, and the ophthalmoscope are shown in Fig. 122.

The surgeon should see the red fundus reflex, and, if the refractive

Fig. 122.—Relative positions in direct ophthalmoscopy.

media are clear, he should by proper focusing be able to find the arteries, veins, macula, and optic disc. If the surgeon has an error of refraction he should wear his correcting lenses; the patient's error can be corrected by means of a lens placed behind the ophthalmoscope. Both the patient and the surgeon should keep both eyes open. The patient should not look directly into the mirror, since this will cause the pupil to contract, in case a mydriatic has not been used. The surgeon should learn to lay aside his accommodation, and this is an art that comes only with practice. The tyro will look at the fundus as at an object very close, while the experienced ophthalmoscopist will view it as if it were far distant. The patient's accommodation should be kept in abeyance either by the use of a cycloplegic or by gazing at a distant object.

In using the indirect method, the surgeon is to hold the mirror with the right hand at 50 centimetres from the patient's right eye, while the strong convex lens is placed in his left hand (Fig. 123). When the left

eye is being looked at, the surgeon should use his left eye and left hand. To see the head of the nerve the patient should turn the eye slightly toward his nose. The macula can be brought into view by having the patient look directly at the mirror. The peripheral parts of the retina can be examined by rotating the eye peripherally. If the view of the fundus is not clear, it can be made so by the use of a strong convex lens. Reflexes from the cornea and lens are often complained of by the examiner. They can be eliminated by tilting the mirror, by changing the position of the lens, or by altering the situation of the light.

THE SIZE OF THE OPHTHALMOSCOPIC IMAGE varies with the conditions under which the methods are employed, and with the refraction of the two eyes. The magnification of the details of an emmetropic eye examined by direct ophthalmoscopy is about seventeen times that of normal. In hypermetropia it is less; in myopia it is greater. A plane mirror gives a larger, but less brightly illuminated, image than the concave one.

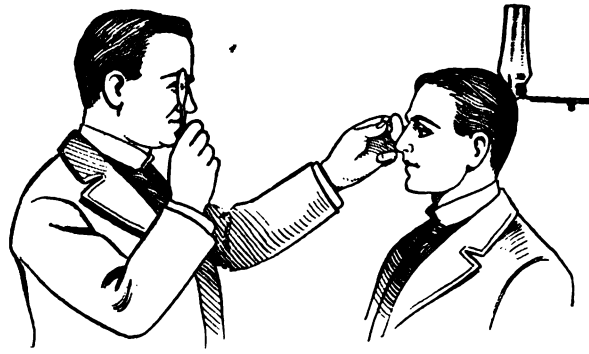


Fig. 123.—Indirect ophthalmoscopy.

USES OF THE OPHTHALMOSCOPE.—The ophthalmoscope is used: (1) to detect opacities in the dioptric media, (2) to study the fundus, (3) to determine the refraction, and (4) to demonstrate differences of level in the fundus.

To Detect Opacities the concave mirror of the ophthalmoscope is used. The opacity appears as a dark cloud or black spot on the red background. Any spot appearing black by this method of examination looks white or gray by oblique illumination, for this reason: rays of light from the fundus, falling on an opacity from behind, are returned unseen by the surgeon; and, in oblique illumination, rays of light striking an opacity from in front do not reach the retina, but are reflected into the eye of the examiner. To locate an opacity it should be remembered that opacities in the cornea and lens are immovable, while vitreous opacities are generally floating, but in rare instances may be fixed. Hence, after moving the eye, vitreous opacities float, while corneal and lenticular ones are stationary. In many cases an opacity can be localized by oblique illumination. To distinguish between

an opacity in the cornea, and one in the anterior or posterior part of the lens, it is often necessary to use parallactic displacement. This can be understood by reference to Fig. 124, in which the points, *a*, *b*, and *c* represent, respectively, opacities in the cornea, anterior and posterior parts of the lens. If the surgeon looks at the eye in the direction of the optic axis, he sees only one opacity, as shown in the upper figure. If, however, the patient looks downward, all of the opacities are seen, that of the posterior part of the lens being highest, that of the anterior part of the lens in the middle, and the corneal opacity lowest. An apparent movement upward when the eye is actually turned downward proves that the opacity is behind the plane of the iris; an apparent movement in the same direction as the actual movement of the eye shows the opacity to be located in front of the plane of the pupil. If the patient's eye is held still and the surgeon moves, the opposite is true, opacities in front of the pupil seemingly moving in

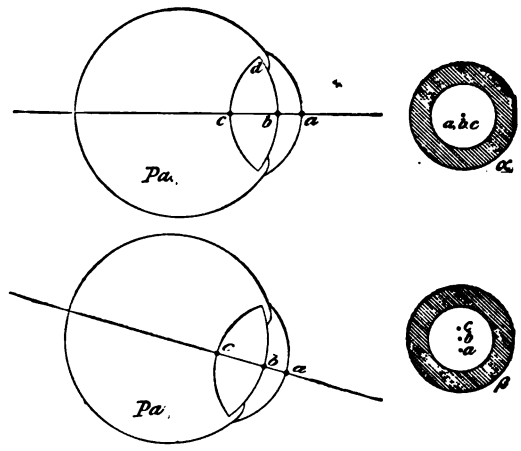


Fig. 124.—Localization of opacities in the lens and cornea. (FICK.)

the opposite direction, while those behind the iris move in the same direction. Finally, any opacity which is found is to be inspected at close range by using a strong convex lens behind the ophthalmoscope. Besides its use in finding opacities, the ophthalmoscope, used as described above, readily detects a tear in the iris or the existence of a partial dislocation of the lens.

Examination of the Fundus.—The media being clear, the surgeon proceeds to examine the fundus, a description of which is found in Chapter II. It will be well to use the indirect method first, thus gaining a general idea of the state of the eye. The surgeon sits about 40 centimetres from the patient, illuminates the pupil with light reflected from the mirror, interposes the convex glass in the path of luminous rays, and searches for the head of the optic nerve. The lens must be held squarely in front of the eye, otherwise the fundus will appear distorted. To find the optic disc, in examining the right eye, the patient should look in the direction of the

surgeon's right ear. After studying the disc for pathologic changes the vessels, the macula, and the peripheral parts of the retina are passed in review. The fundus is next to be examined minutely by the direct method. Inability to see the details of the fundus clearly will suggest the presence of an error of refraction too great for the accommodation to overcome.

The Ophthalmoscope as a Refractometer.—Used as a refractometer, the ophthalmoscope is employed for two purposes: to determine the nature of an error of refraction and its amount. The former is accomplished by holding the mirror at from 30 to 50 centimetres from the eye to be examined. The surgeon illuminates the eye, looks through the mirror, and seeks for vessels. If any are seen, the eye is ametropic. To determine the nature of the error the surgeon has only to move his head from side to side and watch the movement of the vessels. If they move apparently in the same direction as the surgeon's movement, the eye is hypermetropic; if opposite, it is hypometropic (myopic).

In the hands of experts the ophthalmoscope can be used to determine the amount of refraction errors. Either the direct or indirect method may be employed, although the former is preferred as the simpler procedure. In the remarks to follow reference will be made alone to the direct method. In order to measure refraction with the ophthalmoscope, several conditions must be complied with:—

1. The accommodation of both patient and surgeon must be relaxed.
2. A particular part of the fundus must be selected to be refracted.
3. The surgeon must be emmetropic; if ametropic, his error must be corrected.
4. The surgeon should approach as closely as possible to the eye to be refracted.
5. The findings of the ophthalmoscope must be confirmed by other tests before the surgeon prescribes for the patient.

In explanation of these rules it is necessary to state that the accommodation of the patient is usually relaxed by the use of a cycloplegic or by having him look at a distant object, while the art of holding his own accommodation in abeyance comes to the surgeon after long practice. In selecting a part of the fundus to be refracted, it is customary to choose either the optic disc or, preferably, one of the vessels at the temporal side of the disc near the macula. The surgeon should approach a point 13 millimetres in front of the cornea, this being the anterior focal point of the eye. If a greater distance is chosen, it will be necessary to subtract the distance from the glass to the cornea from the lens selected to correct hypermetropia, and add it to the correction for hypometropia (myopia).

In case an emmetropic eye examines another eye which is emmetropic, the image will appear clear and distinct, because rays coming from the normal eye are parallel. In Fig. 125 the examiner, *E*, looks into the emmetropic eye of the patient, *T;* and a clear image of *St* will be formed at *Mo* on the retina of the examiner. If, however, the surgeon does not

obtain a clear image of the fundus, it will be in order for him to place a convex glass behind the mirror.

If this improves the picture of the fundus, the surgeon increases the strength of the glass until he obtains the strongest convex lens through which the details of the fundus can be obtained. This glass is the measure of the patient's hypermetropia. Ophthalmoscopy under these conditions is explained by Fig. 126, in which rays coming from the retina of the hypermetropic eye, *H*, appear as if coming from a more remote point, *I*,

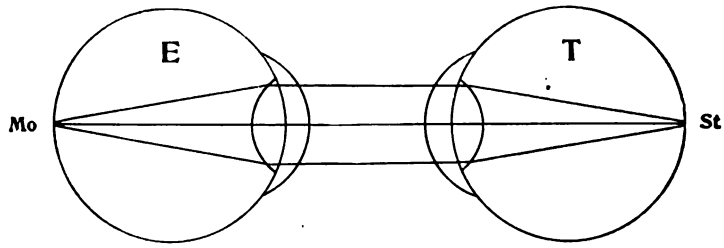


Fig. 125.—Ophthalmoscopy in emmetropia.
The eye of the examiner, *E*, and of the patient, *T*, are normal in refraction.

behind the eye. The convex lens renders the divergent rays parallel, and they are brought to a focus at *C* on the retina of the examiner.

If the surgeon finds that his view of the fundus is made more dim by the convex glass, he should move the disc and place a concave lens behind the mirror. The weakest concave glass with which the details of the fundus can be recognized is the measure of the patient's hypometropia (myopia). Possibly the surgeon will be able still to see the fundus through a glass 1 or 2 D. stronger; but he does so only by calling his accommodation into

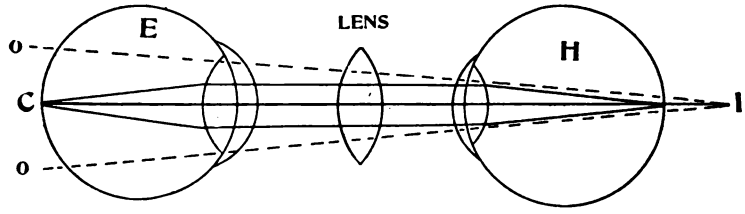


Fig. 126.—Measurement of hypermetropia by direct ophthalmoscopy.

play. Rays emerging from the myopic eye are so convergent that they would meet at the punctum remotum, *C* (Fig. 127). The concave lens renders them parallel, and they fall on the emmetropic eye of the surgeon to form a focus at *s*.

The application of ophthalmoscopy to astigmatism comprises, first, the diagnosis of astigmatism, and, second, its measurement. For these purposes the direct method is preferred. If the surgeon examines an astigmatic eye, he will observe that the picture is blurred in parts; when vessels running vertically are in focus, those taking a horizontal course are blurred,

and *vice versa*. Much stress is laid upon the shape of the optic disc as characteristic of astigmatism, the round disc of the normal eye being seen apparently oval in astigmatism. It is unwise, however, to depend on this. A better criterion is that afforded by the ability of the surgeon to see all parts of the disc clearly at the same time. In astigmatism this will be impossible; when the temporal and nasal sides of the disc are clear, the upper and lower borders are out of focus in the astigmatic eye. The differences produced in the retinal picture by irregularity of curvature of the lens or cornea will enable the surgeon to measure the defect by direct use of the ophthalmoscope. The retinal vessels are taken as the objective points in the examination. It is to be remembered that horizontal lines or vessels are seen through the vertical meridian of the cornea and vertical lines or vessels are seen through the horizontal meridian. Remembrance of this simplifies the problem which, in truth, is the measurement of the hypermetropia or hypometropia (myopia) of each of the principal meridians, the difference between these findings constituting the astigmatism. If, for example, the surgeon sees the vertical vessels clearly with a weak concave lens (2 D.) and the horizontal vessels are clear only after the interposition

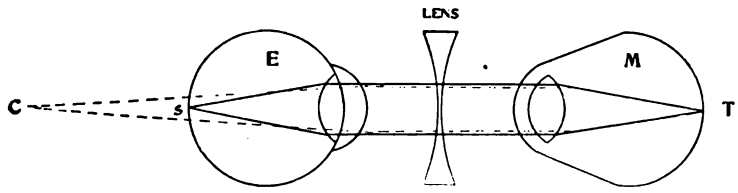


Fig. 127.—Measurement of hypometropia (myopia) by direct ophthalmoscopy.

of a convex glass of 4 D., it follows that the case is one of mixed astigmatism (myopia of 2 D. in the horizontal and hypermetropia of 4 D. in the vertical meridian). If the horizontal vessels are clear without any glass, and the vertical ones are seen best with a convex 1 D., there is simple hypermetropic astigmatism of 1 D. Should the vessels in one principal meridian be clear only with a + 2 D. lens and those in the other principal meridian require a + 3 D. glass, the case is one of compound hypermetropic astigmatism. In the estimation of refraction by ophthalmoscopy the measure of error is the weakest concave lens and the strongest convex one which renders the vessels clear.

In conclusion it must be said that the estimation of refraction by the ophthalmoscope is unreliable, except in the hands of a few experts, and that in all cases the findings by this method should be confirmed by retinoscopy, keratometry, and the use of the trial-case.

The Determination of Differences of Level in the Fundus, which is of practical value in the swelling of the nerve-head occurring in optic neuritis and its excavation in glaucoma, may be accomplished in one of two ways: by the phenomenon of parallax or by the measurement of the refraction of

two points occupying different elevations. Parallactic displacement is produced by moving the convex lens in indirect ophthalmoscopy. By shifting the lens it is noticed that two observed parts of the fundus image move at unequal rates in case a difference of level exists. Thus, in a glaucomatous excavation, the vessels climbing over the edge of the optic disc will move faster and in front of those in the bottom of the cup. In case a projection of the nerve-head occurs, as in papillitis and in intra-ocular tumor, the same phenomenon will be observed. Appreciation of parallactic displacement may be facilitated by study of Fig. 128. Here a glaucomatous excavation of the nerve-head reaches from *o* to *s*. When the convex glass is placed at *B*, the points *o* and *s* are in line, and the idea of depth does not obtain, since one point covers the other. If, however, the lens is displaced to *C*, it will be observed that the image of the point *o* is reproduced at *oo* and that of *s* is likewise reproduced at *ss;* and the points seem to have separated, *o* moving more rapidly than *s*.

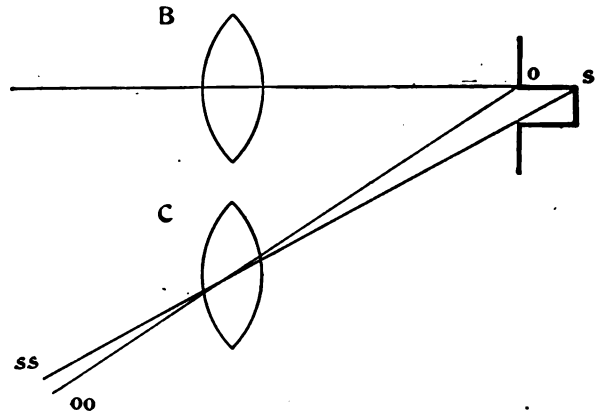


Fig. 128.—Diagram to show parallactic displacement in indirect ophthalmoscopy.

In order to measure elevations and depressions of the fundus by ophthalmoscopy it is necessary to proceed as in the measurement of errors of refraction: the glass is found which brings the depth of an excavation into focus and then it is ascertained what glass will clear the image of the parts normally situated. The difference expressed in dioptres and multiplied by 0.3 will give the depth of the excavation in millimetres. For example, let it be supposed that, in a case of glaucomatous cupping, the depth of the excavation is in focus with a — 5 D. glass, and the margin of the disc and adjacent retina are best seen with + 1 D., a difference of 6 D. The depth of the excavation is 1.8 millimetres. In the same manner a swelling of the nerve-head can be measured.

The Fundus Reflex Test.—If light be thrown into the eye by a mirror, and the mirror be slightly tilted while the surgeon is looking through it, a shadow will be seen passing across the pupillary area. The direction and

rapidity of movement of the shadow are indicative of the kind of refraction error and its amount. A rapidly moving shadow means a small error; a slowly moving one means a considerable or large degree of ametropia. The direction in which the shadow moves tells the skilled observer the kind of error; and the direction of movement depends on whether a concave or a plane mirror is employed. Retinoscopy (skiascopy, the shadow test, koroscopy, keratoscopy, pupilloscopy) is the determination of the refraction by the observation of the shadow which results from reflecting light into the eye by a mirror. In describing this test ophthalmic writers use the terms "area of light," "the illuminated area," and "the illumination" synonymously; while the terms "shadow" or "shade" mean practically the same thing, since the "shadow" is simply the place where the illuminated area ceases and darkness begins. It is not meant that the shadow of any object is thrown on to the retina.

In practical value this test is not surpassed by any. Being an objective test, it is of the greatest importance in the young, the deaf and dumb, the illiterate, among malingerers and plaintiffs seeking damages, and in all other

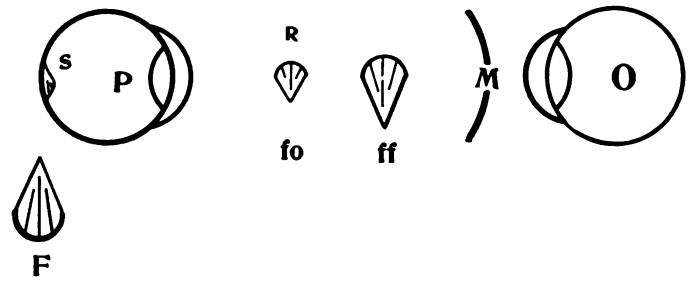


Fig. 129.—Diagram to explain retinoscopy with the concave mirror. (After LANDOLT.)

cases of refraction error. By its use not only can myopia and hypermetropia be measured accurately, but it also enables the surgeon to measure the amount of astigmatism and determine the direction of the principal meridians.

The principle of retinoscopy is the finding of the point of reversal, or far point, of a myopic eye. When the myopic eye is examined by direct ophthalmoscopy an erect image is seen; at some distance from the eye an inverted aërial image appears, which is formed at the conjugate focus, or far point, of the eye. It is here that the erect changes into the inverted image. Retinoscopy is a method of determining this point of reversal. Emmetropic and hypermetropic eyes are to be given an artificial myopic far point by the addition of a convex spheric lens.

The changes occurring in an eye examined with the concave mirror have been explained by Landolt.

In Fig. 129 the surgeon, *O*, examines the eye of the patient, *P*, by the concave mirror, *M*, the source of illumination being at *F*. The mirror

produces a real inverted image of the flame at *ff*, which becomes the actual source of illumination. The observed eye receives a retinal image, *s*, of *ff*, and, regardless of its refractive condition, the image is inverted relatively to the object. The image will be more or less distinct according to the refraction. If the image is inverted its movements also are inverted relatively to those made by the object. If the mirror is rotated from right to left, the real image, *ff*, passes from right to left also, while the retinal image, *s*, moves from left to right. It can be easily demonstrated that, regardless of the refraction of the examined eye, the image of the flame which illuminates the fundus always moves opposite to the motion of the concave mirror.

However, the illuminated part of the fundus becomes the object for the surgeon's eye; and this fact, subject to the law of conjugate foci that the image can replace the object and the object take the place of the image, lies at the basis of retinoscopy, for the retina and far point of the eye are conjugate foci, images being formed at either according to the refraction of the eye examined. If the eye is myopic 1 D., its far point is at 1 metre, and this is the point of reversal. If the eye of the patient is emmetropic or hypermetropic, rays coming from it are not brought to a focus, unless on the retina of the surgeon's eye. Hence, the surgeon sees the fundus in the erect image, and the ocular luster moves in the same direction with the image of the flame at the fundus of the eye, *P: i.e.*, opposite the movement of the mirror. With the myopic eye, however, the case is different. Let us suppose that *P* is a myopic eye whose far point is in the plane of *R*. When such an eye is examined with the concave mirror, an inverted image of *s* appears at *R*, the inversion being produced by the dioptric media of the eye. *R*, therefore, has the same direction as *ff*, and, of course, undergoes the same movements. If *ff* moves with the mirror from right to left, and *s* from left to right, then *R* moves from right to left. It is evident that *s* is simply the illuminated part of the fundus which the surgeon observes in the patient's pupil. The pupil is seen in its real position, while the fundus appears inverted. If the mirror which illuminates the eye passes from right to left, the pupil is lighted up likewise from right to left, although, in reality, the light passes from left to right at the fundus of the examined eye. Hence in myopia the shadow moves in the same direction as the concave mirror. This, however, is true only when the surgeon is at a certain distance. If the surgeon approaches *P* closely enough to intercept rays before they reach their focus, as occurs when *O* is between *R* and *P*, the inverted image is not formed in the air in front of the surgeon, but only in his eye, as in emmetropia or hypermetropia. In this case the shadow would move the same in myopia as in emmetropia and hypermetropia. Consequently the refractive condition cannot be diagnosed at the first glance by retinoscopy. If the surgeon is 1 metre from *P*, the inverted image cannot be seen unless the myopia of *P* is more than 1 D. If the surgeon is slightly more than 1 metre from *P* and the examined eye shows a general

illumination without any movement of a shadow, we know there is myopia of 1 D. If the shadow moves rapidly against the mirror when *P* is examined, and after adding a + 1 D. lens the shadow does not move, but the whole pupil is brilliant, we have to deal with emmetropia. If a convex 4 D. lens is needed to produce such a result, we know the amount of hypermetropia is 4 D. — 1 D. = 3 D. If a concave 4 D. lens is required, the amount of myopia is 4 D. + 1 D. = 5 D.

Thus we have seen what is the real movement of the light on the fundus, and this can be demonstrated in an enucleated eye; however, it is not this, but the apparent movement with which the surgeon is concerned. With the concave mirror the apparent movement in the pupil is the same as the real movement in the fundus in emmetropia, low myopia, and hypermetropia. In myopia of more than 1 D. it is the opposite: *i.e., with* the mirror.

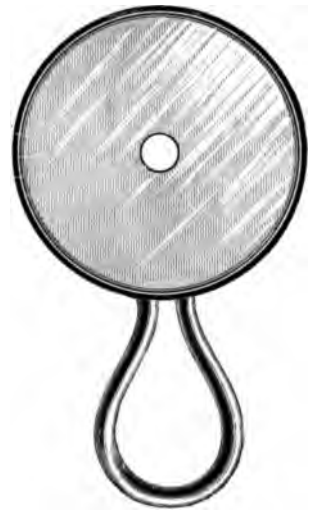


Fig. 130.—Retinoscopy mirror.

In using this test the surgeon should wear his correcting glasses, if he be ametropic. He need not give attention to his accommodation, but should keep both eyes open and observe closely the movement of the shadow. The patient's accommodation, however, must be eliminated by means of a reliable cycloplegic. To refract the macula, the patient should look at the sight-hole of the mirror. The instruments needed for retinoscopy are a concave mirror of 2 centimetres' diameter and 25 centimetres' focus, with a circular central perforation 2 millimetres in diameter; a movable gas-bracket with an Argand burner; an asbestos chimney with iris-diaphragm (Fig. 131); a trial-frame and box of trial-lenses; and a room that is perfectly dark. The remarks to follow concern retinoscopy with the *concave* mirror.

The light is to be placed above and behind the patient's head, the surgeon sitting 1 metre from and directly in front of the patient. Light from

the mirror is to be thrown on to the eye to be examined. The surgeon, looking through the mirror, moves it slightly in a vertical, then in a horizontal, direction, and notes the movement of the shadow. With the concave mirror the shadow moves *with* the mirror in myopia greater than 1 D., and *against* the mirror in emmetropia, hypermetropia, or low myopia. Let us

Fig. 131.—Thorington's light-screen with iris-diaphragm.

suppose that the shadow moves slowly against the mirror. This means that the case is one of hypermetropia of considerable amount. The trial-frame is adjusted and the eye not under examination is covered with an obturator. The patient is told to look at the surgeon's forehead; a convex glass (+ 4

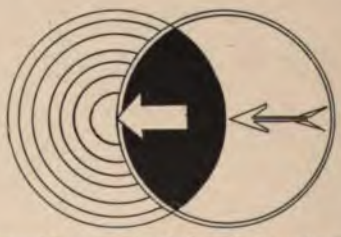


Fig. 132.—Movement of the shadow in myopia.

The concave mirror is used. The arrow at the right shows the direction in which the mirror is moved, and the white arrow marks the direction of movement of the shadow. The shadow moves with the mirror.

D.) is placed in the trial-frame and the shadow is now found to move more rapidly against the mirror. This means that part of the hypermetropia has been corrected. A stronger convex glass (+ 5 D.) is added, which gives not only no motion against, but a slight movement *with*, the mirror. This glass is the measure of the hypermetropia plus the algebraic quantity — 1

D., which must be added because the surgeon is distant 1 metre. Hence, the true amount of hypermetropia in this case is (+ 5 D. — 1 D. = + 4 D.) 4 D. If the surgeon sits 2 metres distant, the algebraic quantity — 0.50 D. must be added to the glass which causes a reversal of the shadow. In using the shadow test the refraction of the vertical meridian is determined by moving the mirror on its horizontal axis; and the horizontal meridian is measured while tilting the mirror on its vertical axis.

Let us take another case: the shadow moves slowly *with* the mirror. This means myopia, and to measure it the surgeon places concave glasses of increasing strength in the trial-frame until the shadow moves slightly against the mirror. This glass, plus the algebraic quantity — 1 D., is the measure of the myopia.

In astigmatism the pupillary area is occupied by a band of light which, as a rule, runs either vertically or horizontally, but may occupy an oblique meridian. Whatever the direction of the band, it indicates accurately the axis of one of the principal meridians. The band of light can usually be

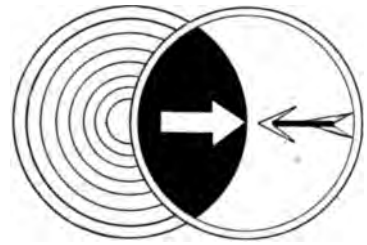


Fig. 133.—Movement of the shadow in emmetropia, low myopia, and hypermetropia.

The concave mirror is used. The arrow at the right shows the direction in which the mirror is moved, and the white arrow marks the direction of movement of the shadow. The shadow moves against the mirror.

seen before a correcting sphere is placed before the eye; but in low degrees of astigmatism the band is noticed only after the proper spheric correction of the myopia or hypermetropia has been made. The problem of measuring astigmatism by skiascopy simply resolves itself into the measurement of the meridian of greatest and that of least refraction, correcting each by the proper sphere. The difference between these findings is the amount of astigmatism. It must be remembered, however, that the edges of the band of light are at right angles to the meridian tested. Thus, in Fig. 135, the diagram at the left (1) shows an astigmatism which is to be corrected by a cylindric lens at 90°, the meridian tested being horizontal, *T;* and the figure at the right (2) shows a similar condition, the axis here being at 180° and the meridian tested being vertical, *To.* As an example of the correction of astigmatism by retinoscopy let us suppose that Fig. 134 represents an eye in which the vertical meridian is neutralized by a + 4 D. lens and the horizontal meridian requires — 4 D. Here is an

instance of mixed astigmatism, and the refraction is expressed in this way: $\begin{matrix} -5.00 \\ +3.00 \end{matrix}$ following the rule that the algebraic quantity — 1 D. must be added to the lens causing reversal of the shadow. The correcting glass in this case can be written in three ways: 1. — 5.00 cylinder (axis, 180°), combined with (⌒) + 2.00 cylinder (axis, 90°). 2. — 5.00 spheric ⌒ + 8.00 cylinder (axis, 90°). 3. + 3.00 spheric ⌒ — 8.00 cylinder (axis, 180°). The third way is used by most opticians, who prefer working minus cylinders on to plus spheres.

The obtaining of the exact axis in astigmatism is much facilitated by retinoscopy. For this purpose a meridian indicator, called an axonometer (Fig. 136), can be placed in the trial-frame and rotated until the white lines on either side of the central opening coincide with the long axis of the band of light seen in the pupil. The degree on the rim of the trial-frame, to which the axonometer points, is the axis at which the cylinder is to be placed.

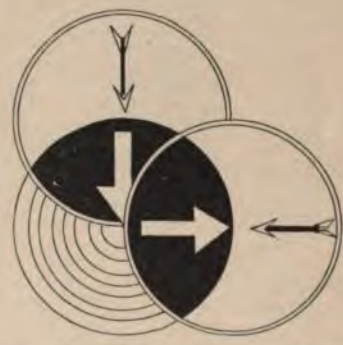


Fig. 134.—Movement of the shadow in mixed astigmatism.

The direction of the (concave) mirror movement is indicated by the black arrow; the white arrow shows the direction in which the shadow moves.

The overcorrection of retinoscopy depends on the distance between the surgeon and the patient. When this is 1 metre the overcorrection equals the focal length of a 1 D. glass; if at 2 metres, 0.50 D.; and at 4 metres, 0.25 D. Most ophthalmologists use retinoscopy at 1 metre; some, however, hold that the detection of a low degree of astigmatism becomes easier at 2 metres. This amount is to be subtracted from the skiascopic correction, thus giving the actual amount of refraction-error. This finding, however, is not the measure of the glass to be prescribed, since ophthalmic surgeons find that it is inadvisable to prescribe the full correction of an error. The amount of reduction will be fully discussed in the chapter on refraction. In any case the retinoscopic findings should be confirmed by the use of the trial-case and the ophthalmometer.

Retinoscopy with the Plane Mirror.—With the plane mirror it is necessary to have the source of illumination close to the mirror. The

movement of the shadow with the plane mirror is the reverse of what obtains when the concave one is used, viz.: with the plane mirror the shadow moves *with* the mirror in emmetropia, hypermetropia, and low myopia. The retinoscopic examination is much facilitated by the use of one of the several skiascopes which are on the market. These are instruments holding lenses and permitting the rapid change of the lens-strength. Some of them are to be held by the patient, while others are turned by the

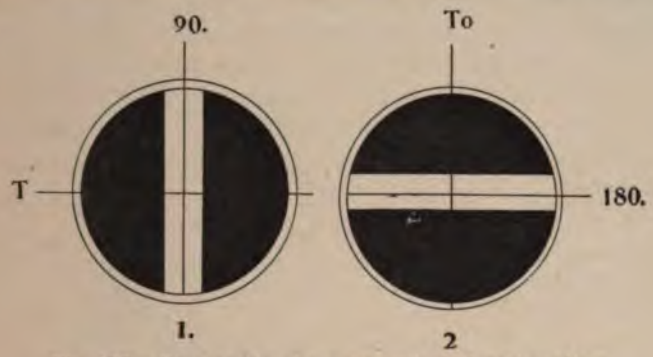


Fig. 135.—Diagram to show correction of astigmatism.

surgeon. Among the hand-skiascopes are those of Snell, Gruening, and Marple; revolving skiascopes have been invented by Jennings, Lambert, and others.

Tests for Simulated Blindness.—The tests for simulated blindness are the obstruction device, various tests with prisms and lenses, the mirror test, the stereoscopic test, Snellen's colored letters, and Hering's experiment with

Fig. 136.—Thorington's axonometer.

falling bodies. These tests apply to simulation of blindness in one eye. If the patient claims blindness in both eyes only careful watching will expose the deception.

The Obstruction Device can be employed by holding a ruler on the flat in front of a book from which the patient reads. If binocular vision exists, he will see all the words and will read without halting; if one eye is blind, certain words will be hidden by the ruler.

Tests with Prisms are numerous. The following may be used: 1. Place a 7° or 8° prism, base up or down, in front of one eye and direct the patient to look at a candle at 6 metres. If he sees double, binocular vision is present, and he is shamming. 2. Place in front of each eye a 7° prism, base out, and ask the patient to look at a flame at 6 metres; if binocular vision exists there will be a noticeable convergence of the visual axes. 3. Hold a 12° prism, base out, before the blind eye. If the eye sees there will be a movement of it inward to overcome crossed diplopia. 4. Place a square prism with its edge half-covering the pupil of the good eye, the supposedly blind one being covered. Monocular diplopia is thus produced. Now move the prism entirely over the good eye and at the same time uncover the other; if diplopia still exists, the patient is malingering. 5. Place a prism, base vertical, in front of either eye and direct the patient to walk downstairs. If he closes one eye binocular vision is present. 6. Place a piece of Iceland spar in front of one eye. If three images are present, binocular vision exists. 7. Place a Maddox double prism accurately before one eye, the other being uncovered. The presence of three images proves binocular vision. 8. Place a + 14 D. glass before the sound eye and a — 0.25[s] before the other and ask the patient to read test-types at 6 metres. If he reads, it is with the eye which he claimed was useless—barring, of course, aphakia. 9. Place the Maddox rod before the good eye. If the patient sees both the flame and the light-streak, he is shamming. 10. Use Snellen's spectacles and red-green test-letters. The spectacles have one red and one green glass. The patient is to read the transparent red and green letters, which are hung up in a window. It is best to have him read the letters first without the spectacles. Then the spectacle-frame is adjusted, both eyes being kept open, and the letters are to be read. If the patient does this, binocular vision is present. The green glass excludes all the rays from the red letters and likewise the red glass excludes rays from the green letters. Hence some letters are seen only with one eye, and others with the other.

Hering's Experiment with falling bodies can be used as a test for malingering. The patient looks with one eye through a long tube at a thread stretched vertically. Glass beads are then dropped in front of and behind the thread. If binocular vision is present the patient will correctly state whether the beads were in front of or behind the thread; if blind in one eye he will make numerous mistakes.

Stereoscopic Tests are numerous. One of the simplest is to use the letters L and F, which, to the person with binocular vision, make E. If the patient says he sees E, he is using both eyes.

Fridenberg's Mirror Test is of great value. It consists in the use of an instrument carrying a mirror and two test-cards, and is described as follows: "The mirror is mounted on a horizontal arm in such a way as to permit of varying its distance from the test-card, and of presenting it alternately to either eye by revolving the bearing through an arc of 180°.

The lateral tilt of the mirror can be changed at will, and is indicated by a pointer on a horizontal scale. When the pointer is at 90°, the plane of the mirror is at right angles to the line of vision of the eye on the corresponding side, and this eye sees its own image. The test-card on this side, however, is not normal to the mirror, and its reflection is seen only by the opposite eye, which the subject presumes to be unconcerned in the visual act, as it does not appear in the mirror.

"A slight tilting of the mirror to the temporal side, bringing the pointer to 95° or 100°, is sufficient to reverse the optical conditions, so that the test is seen only by the eye on the same side. By switching the mirror over to the opposite side of the arm, a similar double test can be applied, so that, in all, eight variations are rapidly obtained, as follows:—

Fig. 137.—Test for simulated blindness. (FRIDENBERG.)

MIRROR BEFORE RIGHT EYE.	MIRROR BEFORE LEFT EYE.
95° R. E. sees right card.	95° L. E. sees left card.
90° L. E. sees right card.	90° R. E. sees left card.
70° R. E. sees left card.	70° L. E. sees right card.
60° L. E. sees left card.	60° R. E. sees right card."

Localization of Foreign Bodies.—Occasionally a foreign body can be seen by oblique illumination or by ophthalmoscopy. In most cases, however, it is out of view either because of its position, the presence of blood, distortion of tissues, or inflammatory changes. In such cases attempts were formerly made to locate it, if of iron or steel, by the movement of magnetic needles arranged in the sideroscope of Asmus or of Hirschberg. The attempt has been made to accomplish the same result by bringing strong magnets in close proximity to the eye, expecting the movement of the foreign body to produce pain, and thus assist in the localization, which may be the case if the foreign body is jagged in outline and situated in the ciliary region. This method has been superseded by the use of the x-rays. Non-metallic, as well as metallic, particles can be located in almost all

cases. The methods of greatest value in localization by the x-rays are those of Leonard, Sweet, and Davidson. All are founded on triangulation.

Leonard's Method.—Leonard, who was a pioneer in the use of the x-ray in ophthalmic surgery, devised a method of localization in which the base-line for triangulation is made anterior to the cranial shadow. The exposures are to be repeated at fixed distances and set situations, giving a series of relational sides and angles from which the surgeon calculates the position of the foreign body.

Sweet's Method.—Sweet fixes to the side of the patient's head a plate-holding device, which carries two steel rods, each bearing a ball at its end. These ball-pointed rods are at all times parallel to each other and with the photographic plate, and are at a known distance apart. One rod points to the centre of the cornea, the other to the outer canthus, both being parallel to the visual line and perpendicular to the plate. Two radiographs are made, one with a tube parallel, or nearly so, with the plane of the two rods, and the other above or below this, the first situation. From a study of the shadows cast by these ball-pointed rods in relation to the shadow of the foreign body, the surgeon is able, on a horizontal and vertical diagrammatic section of the eyeball, to locate the position of the foreign body. This is achieved by taking the distance that the shadow of the foreign body is above or below the shadow of the indicating balls on the two negatives, and transferring these measurements above or below the spots representing the two indicator balls on the vertical section of the eyeball. A line drawn through these points gives the plane of shadow of the foreign body at the two exposures, their crossing point indicating the situation of the metal in the eye as studied in relationship to the centre of the cornea.

In determining the position of the body when back of the centre of the cornea, the distance is measured so that the shadow of the body is posterior to the shadows of each of the two balls on the plate made with the tube horizontal to the plane of the indicators. These measurements are entered above the spots representing the two balls on the diagram of the horizontal section of the eyeball, and a line is drawn through these points which indicates the plane of shadow of the foreign body. A line is now drawn perpendicular to the situation of the body as found on the vertical section of the ball. Where it crosses the line representing the plane of shadow on the horizontal section is the situation of the body back of the anterior portion of the cornea.

Davidson's Method.—Mr. McKenzie Davidson uses a Crookes tube whose anode is made of osmium (so as to obtain the best definition) placed between two pieces of platinum, a head-clamp, and a localizer. Two sciagraphs are taken from different points of view. First, a piece of lead wire, 1 centimetre long, is plastered on to the patient's eyelid, forming a fixed point in the picture to be taken. The position of the tube can be moved by means of a crossbar. The side of the head with the injured eye being fixed flatly against two piano wires stretched across a space in a board

admitting a photographic plate, as shown in Fig. 138, the first picture is taken; then the bar is moved 6 centimetres and the second picture is made. The patient meanwhile looks at a point directly in front, so situated that the visual axis of the injured eye is parallel to the horizontal wire. On the negative are seen the images of two wires which cross at right angles. They are stretched across the window in which the photographic plate is fastened. The two negatives are fixed and each one shows the lead wire and the foreign body. After developing the negatives, a tracing in celluloid is made of each, showing the crosswires and any foreign body present. By

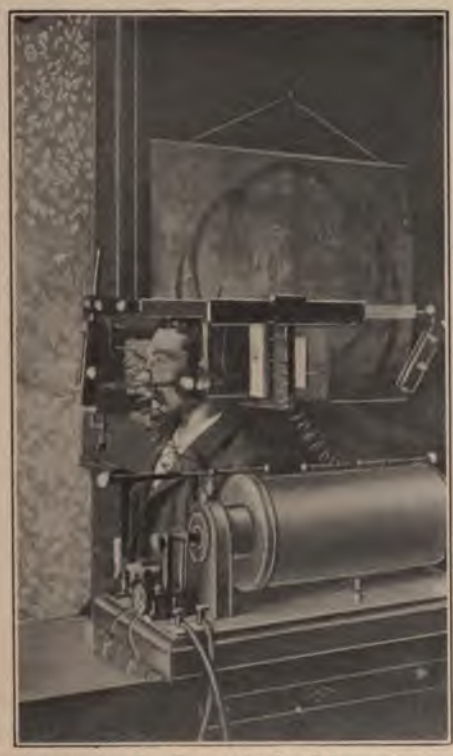


Fig. 138.—Apparatus for fixing the head in x-ray examination. (DAVIDSON.)

mathematical calculations it would be a tedious task to locate the foreign body; hence Mr. Davidson has designed his localizer, which consists of a stand with a plate-glass top under which is a mirror for throwing light from below upward. A horizontal bar, on which is a millimetre scale, projects from the top of the table. On the glass plate are two lines cut at right angles, corresponding to the two crosswires shown on the negative. The sliding millimetre scale is placed above the glass plate a distance equal to the distance of the anode of the Crookes tube from the negative at the time the picture was being taken. At the time of the exposure the surgeon should mark on the patient's skin the quadrant which corresponds to the

lead wire. (The crosswires have previously been brushed over with ink or a suitable dye and the patient's head, resting on the wire, is marked into quadrants by the dye.)

The negative is now placed on the localizer. Since the x-ray is not bent, but passes straight through an object, it is possible to represent the path of the ray by threads stretched from the scale to the image of the foreign body on the negative. This is shown in Fig. 139. The further procedure is thus described by Mr. Treacher Collins:—

"The negative with the two images of the foreign body and of the pointing lead wire, when developed, is placed on the stage of an instrument called 'the crossthread localizer.' It resembles a photographer's re-

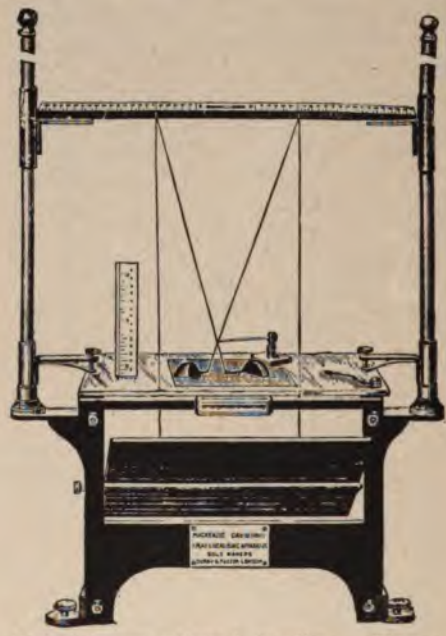


Fig. 139.—The crossthread localizer. (DAVIDSON.)

touching desk. Upon an iron stand a piece of plate glass is placed horizontally and level. Below this is a mirror, which can be adjusted so as to reflect upward the light from a window or lamp. Above the plate-glass stage is a horizontal bar with a millimetre scale which slides up and down upon two vertical brass rods. The plate-glass stage has two lines cut at right angles to each other on it; when the instrument is in use the horizontal bar is made to bear the same relation to these cut lines as the horizontal bar, along which the Crookes tube moved, did to the crosswires.

"When the negative is laid on the stage, the white cross on it produced by the wire is made to lie on the cut lines; so that it is placed in relation to the horizontal bar under exactly the same conditions as it occupied when it was being taken.

"The path of the x-rays during the two exposures is traced by means of threads, which are separated on the horizontal bar the same distance as that which separated the position of the tube during the two exposures.

"Small lead weights with needles attached, through the eyes of which the threads are secured, are used to keep the threads taut, and direct them to the exact points on the negative required.

"If the two threads are arranged to start from the horizontal bar in the positions in which the tube was during the two exposures, and the point of each needle carrying the thread is placed on a corresponding point of the image produced by each exposure, then the position where the threads cross will correspond to that occupied by that point of the foreign body in relation to the plane of the plate. The distance is next measured from the two vertical planes represented by the shadows of the crosswires. This is done by the use of an upright square placed with its edge coincided with the edge of the shadow, first on one side of the foreign body and then on the one at right angles. The relation of the pointing lead wire is also estimated to these planes and to the foreign body, and with these data the exact position of the foreign body in a schematic eye is easily worked out."

THE SIDEROPHONE.—Jansson has invented an instrument for the detection of pieces of iron or steel within the eye. It consists of an electromagnet to which a telephone is attached. When brought near a piece of iron a change is produced in the electric current, and this produces a sound in the telephone which can be easily heard.

CHAPTER V.

DISEASES OF THE EYELIDS.

The eyelids are subject to congenital anomalies, tumors, traumatisms, inflammations, parasites, degenerations, infiltrations, etc. Owing to the complexity of the structures entering into their formation, the lids show many symptoms pertaining to general diseases. The diseases of the conjunctiva are of such importance as to demand consideration in a separate chapter.

CONGENITAL ANOMALIES OF THE EYELIDS.

These defects are rarely met with. The following conditions have been found:—

Ablepharia.—Partial or complete absence of the lids may occur on one or both sides, and is a rare defect.

Coloboma of the Lids generally is present as a triangular defect with the base toward the lid-margin and the apex directed toward the margin of the orbit. It is more frequent in the upper lid. Cases in which the coloboma is situated at the inner or outer canthus are extremely rare. The defect commonly involves the entire thickness of the lid, and in extent varies from a mere notch to a deep fissure. In the colobomatous area Meibomian glands and cilia are absent. In some instances the defect is filled by a bridge of tissue which connects the lid with the globe or with the surface of the cornea. In others it is associated with dermoids of the corneoscleral region or with subconjunctival lipomas. In still other cases corneal opacities or pterygium-like formations are present (von Hippel).

Cryptophthalmos.—Under this name Zehender and Manz have described a congenital condition in which the chief characteristic is the passage of skin over the base of the orbit, completely hiding the eyeball. The name is incorrectly applied to a similar condition of the skin with absence of the globe. In most cases of cryptophthalmos other defects are present. The affection was bilateral in six and unilateral in three of the reported cases. Cilia, eyelids, and conjunctival sacs were absent. The eyebrows were present in two cases. Perception of light is present in cryptophthalmos, as is evidenced by contraction of the skin over the globe when light is concentrated on the base of the orbit. The condition is irremediable.

Symblepharon.—This is a cohesion between the eyelid and the eyeball, and may be partial or complete. In cryptophthalmos there is complete obliteration of the conjunctival sac. Partial adhesion of the lid to the globe is sometimes seen as a congenital condition.

Ankyloblepharon, union between the lid-margins, may be total or partial. A few cases have been observed in which at birth a filiform band passed from one lid to the other.

Blepharophimosis, an adhesion of the lids at the outer canthus, producing a narrowing of the palpebral opening, may occur congenitally. The author has observed it as an hereditary characteristic.

Microblepharon, a congenital shortening of the lids, has been observed by Fuchs and others.

Entropion, a turning in of the eyelid, when congenital is generally associated with other anomalies, such as distichiasis, epicanthus, anophthalmos, or microphthalmos.

Ectropion, a turning out of the lid, when congenital is often found in association with hydrophthalmos, ptosis, microphthalmos, epicanthus, or anophthalmos.

Distichiasis, the presence of rows of supplementary cilia, has been studied by Kuhnt, who found that in lids otherwise normal a second row of very fine hairs grew from the posterior part of the intermarginal space. Study of sections of the lid showed the entire absence of Meibomian glands, the abnormal cilia occupying their places. The glands of Moll and Krause were consequently uncommonly well developed. According to Westhoff, congenital distichiasis is sometimes hereditary.

Epicanthus.—This is a condition in which relaxed folds of skin at the root of the nose extend vertically toward the eyebrows. In pronounced cases they conceal the inner canthus, caruncle, and in some instances the inner half of the lid, giving the appearance of strabismus. It is almost always bilateral and often is associated with ptosis. In Mongolians it is a normal condition. A small amount of deformity existing at birth will often disappear in a few years with the development of the nose. When the skin of the root of the nose is lifted up, the deformity disappears: an observation which caused von Ammon to devise his operation for epicanthus. A spurious form of epicanthus is acquired by syphilitics with saddle-noses. The treatment of epicanthus is chiefly surgical. Broekaert and others have reported successful results from the subcutaneous injection of paraffin, which is molded into the desired form and heightens the nasal bridge.

Congenital Ptosis, a drooping of the upper lid, may be unilateral or bilateral, and often is associated with epicanthus, paralysis of the ocular muscles, or other congenital anomalies. It may be due to redundancy of tissue or to deficient development or absence of the levator muscle. Ptosis should be corrected by operation.

Fistula of the Upper Lid.—This is an extremely rare condition due to non-closure of the fronto-maxillary fissure. One case has been described by Lannelongue and Ménard.

Treatment of Congenital Anomalies.—The treatment of these conditions is entirely surgical, and does not differ from that applied to similar acquired affections.

SKIN DISEASES OF THE EYELIDS.

The skin diseases of the eyelids are numerous. No attempt will be made here to properly classify them. A short description of the principal ones will be sufficient for the purposes of the ophthalmologist.

Erythemata.—The erythematous affections of the eyelids are similar to those which involve other portions of the integument. They properly belong to the domain of the dermatologist, but frequently fall into the hands of the oculist on account of the concomitant conjunctivitis which requires his attention. The one most frequently seen is

Erythema Simplex, an active hyperemia whose causes are various and which disappears upon their removal. This active hyperemia is best combated by cooling evaporating lotions. It is easily recognized as a diffuse flush, of a rather bright color, and is to be distinguished from erysipelas, which is a specific inflammatory trouble. A form of passive hyperemia, sometimes observed, is that due to venous stasis. Here the lids have a bluish-purple hue. The cause of the stasis must be sought and corrected, or a greater or less amount of chemosis will result.

Urticaria (Hives) is an inflammatory disorder of the skin, often involving the eyelids. It is characterized by the presence of wheals with a sensation of itching and burning. In the vast majority of cases it is of gastric origin. It has been seen to follow upon eyestrain or upon improperly corrected errors of refraction (Oliver). The disease should be treated by removal of any source of irritation. The use of an antacid, with proper regulation of the diet, will usually give relief. The chronic form, known as *cnidosis,* should be treated with pilocarpin, quinin, and bromids internally. It may continue for years in spite of treatment.

The inflammatory skin affections of the lids are not so easily allayed, and constitute quite a large class. They are often prone to be obstinate, and local treatment alone will not suffice, but must be combined with internal measures. The most commonly observed of this class is perhaps eczema in its different stages.

Eczema of the Eyelids occurs both in children and in adults, in connection with eczema of the face.

In children it is the rule that the lids are not involved. Infantile eczema assumes three forms: the neurotic, the seborrheic, and the strumous. Neurotic, or reflex, eczema arises from gastroenteric disturbance, and is seen in otherwise healthy, well-nourished children. While the forehead, cheeks, and chin may be bleeding, a trefoil, which includes the orbits, nose, and mouth, is exempt. This is the most frequent type of infantile eczema. In the seborrheic and strumous types the eyelids are often involved. In the latter variety fissures are often present about the canthi, as well as at other mucocutaneous junctions.

In adults eczema of the lids is almost invariably of the erythematous type. Eczema of the lids may show papules, scales, or pustules. All forms itch. Conjunctivitis is often present.

TREATMENT should be adapted to the variety and stage of the disease. Care should be taken to avoid irritating applications. To prevent agglutination, an ointment of the benzoated oxid of zinc may be applied to the lids at night. The conjunctivitis should receive appropriate treatment. The habits and diet should be regulated. In the strumous form of eczema the syrup of the iodid of iron with codliver-oil should be administered. In the seborrheic form the diet should be regulated and a sulphur ointment should be applied locally. The concomitant skin affection should be treated by a competent dermatologist. Otherwise repeated reinfection of the lid-margins will occur.

Fissures of the External Canthus (Rhagades) are often present in cases of eczema and in types of ocular disease causing photophobia and blepharospasm, viz.: conjunctivitis and keratitis. The raw spots should be touched with nitrate of silver, either in the form of a strong solution or the solid stick. In obstinate cases canthotomy may be required.

Impetigo of the eyelids is uncommon, and manifests itself as pustules of a split-pea size, which disappear spontaneously in a few weeks. There are no subjective lesions. Tonic treatment and antiseptic applications are sufficient.

Furuncle.—This form of localized inflammation of the skin and subcutaneous tissue, due to infection by one or more of the pus-cocci, is occasionally seen on the eyelids. The local use of an ointment of salicylic acid (gr. xv to ℥j), and the occasional application of cloths wrung out of hot water, will be appropriate. In neglected cases an incision will be necessary.

Frambesia (Yaws; Amboyna Button; Pian).—This is a contagious disease met with in tropical climates. It is characterized by the presence of raspberry-like nodules in the skin and by more or less constitutional disturbance. The papules undergo suppuration and scabbing, with the formation of a slight scar. In some instances there is serious ulceration of the skin and subcutaneous tissues. The entire course of the disease occupies several months. The eruption, which begins on the face and extends downward, may involve the eyelids, leading to localized thickening, conjunctivitis, and sometimes necrosis. Yaws is to be differentiated from small-pox and the lesions of hereditary syphilis. Its marked resemblance to blastomycetic dermatitis has been recently noted. The prognosis is generally favorable. The treatment includes improved hygienic surroundings, tonics, diaphoretics, and local applications of carbolic-acid lotion or the diluted nitrate-of-mercury ointment.

Furunculus Orientalis (Aleppo Boil; Delhi Boil; Biskra Button).—These names indicate a local infectious disease endemic in the tropics, and characterized by the formation of papules, nodules, scabs, and punched-out ulcers. The uncovered parts of the face are chiefly attacked. The disease is inoculable in both men and animals. Laveran attributes its spread to flies. It is to be distinguished from yaws, which presents marked constitutional symptoms, and is found almost entirely among the colored races.

The oriental boil is a local disease, without constitutional disturbance, and occurs among all tropical races regardless of color. The prognosis is favorable. The treatment includes the use of the galvanic cautery in the early stage; hypodermic injections of 10-per-cent. carbolic solution around the boil; and curettement or the application of caustics, when the process has gone on to suppuration and the formation of granulations. The eyelids are often involved in this disease.

Malignant Pustule (Anthrax Pustule).—This disease, due to inoculation with the anthrax bacillus, may involve the eyelids. It is transferred to man from infected animals, and is found among persons who are employed in the care of animals and among those who handle hides and wool. The infection is carried by the hands, by dirty instruments, or by the bites of insects which have fed on the cadavers. The disease, which fortunately is rare in this country, begins as a small vesicle surrounded by an areola. Soon a livid-red papule develops, followed by a bulla or pustule, and this, in turn, by a black eschar. There is a broad area of edematous infiltration of a violet color. The gangrene may spread rapidly and terminate fatally, or a localized slough may be thrown off, leaving an ulcer, which heals by granulation. When the eyelids are involved there may be extensive destruction, only the ciliary margins remaining. The constitutional symptoms are those of sepsis. The diagnosis will rest on the history of the case, the appearance of the lesions, and the finding of the anthrax bacillus. The prognosis is serious, from 30 to 50 per cent. of the cases ending fatally. The treatment will embrace general supportive measures, incisions, the use of the galvanic cautery to destroy the infected area, and the application of bichlorid solutions. After recovery a plastic operation may be required. Where there is extensive loss of tissue, it will be advisable to suture the marginal portion of the lid to its fellow to prevent lagophthalmos and ectropion.

Carbuncle, an acute phlegmonous inflammation, circumscribed, but more extensive than that of furuncle, may occur upon the eyelids, and is attended with sloughing of the tissues and gangrene of the skin. It should be treated by multiple incisions, curettement of the sloughing material, and packing with gauze. In the treatment care should be taken lest injury be done to the eyeball. A canthotomy may be needed to relieve pressure on the globe.

Abscess of the Lid may occur as the result of injury or orbital disease or from causes not well understood. It should be treated by incision parallel with the fibres of the orbicularis muscle.

Tarsitis, inflammation of the so-called tarsal cartilage, occurs chiefly in syphilitic subjects, and is characterized by great thickening of the lid-margin, often producing considerable deformity. Acute tarsitis is associated with conjunctivitis, blepharitis, and sloughing of the tissues. It is found also in scrofulous subjects. Syphilitic tarsitis occurs as a tertiary symptom. The upper lid droops from increased weight; in tarsitis of the lower lid

ectropion is not uncommon. The treatment includes local cleanliness and the application of remedies suitable for blepharitis, such as ointments of the yellow oxid of mercury, ointment of ammoniated mercury, etc. Internally antisyphilitic remedies are to be used.

Erysipelas.—Although the eyelids are often involved in erysipelas, the disease rarely begins in them. The lids show a dusky-red color; they are edematous and soft; there is great enlargement, the swelling extending over the eyebrow and cheek; the skin presents numerous small blisters, and chemosis and conjunctivitis are often present. By extension into the orbit the disease may cause exophthalmos, atrophy of the optic nerve, and even meningitis and death. The general and local treatment is the same as for erysipelas located elsewhere. Local applications of ichthyol are valuable.

Herpes Zoster Ophthalmicus (Herpes Frontalis) is a form of herpes which, by reason of the ocular symptoms, is of interest to the ophthalmic surgeon. While the disease occurs most frequently in elderly and feeble patients, it is not rarely observed in children and in young subjects with apparently unimpaired nutrition. The initial symptoms include severe pain limited to one side of the head and face, rise of temperature, anorexia, chills, and, in some cases, bronchitis. After the pain has been present for from one to three days unilateral involvement of the skin is apparent along the course of the branches of the first (ophthalmic) division of the fifth nerve, the second and third divisions being rarely involved simultaneously. The eruption begins with groups of papules, rapidly changing into vesicles which vary in size from a pin's head to a split pea. These are surrounded by a bright-red areola. They rapidly become cloudy and form dry crusts. The vesicles occur in patches of three or more, having a tendency to group in a round form. They appear in the temporal region, on the forehead, upper lid, conjunctiva, and cornea. The lower eyelid and cheek are rarely involved in the eruption. The cheek, however, may be edematous by contiguity. The cornea is not always involved in ophthalmic herpes. Many years ago Hutchinson observed that the cornea is involved only in case the nasal nerve is affected. After a variable time the skin heals, leaving pits and depressions resembling those of small-pox. From the peculiar grouping of the scars, it is possible to recognize the disease long after its subsidence. Early in the disease the cornea becomes cloudy; often a large bleb forms, and this, on breaking, leaves an ulcer which frequently causes a scar. Iritis, conjunctivitis, iridocyclitis, and even panophthalmitis are sometimes observed in these cases. Barrett has reported two cases in which diplopia was present. Tension is often increased in the acute stage of the disease and is subnormal in the later period. Rarely ophthalmic herpes is a cause of sympathetic ophthalmitis. Long after the acute symptoms have disappeared the eyeball remains anesthetic, although subject to attacks of neuralgic pain, and the skin feels numb and stiff like parchment. The prognosis is serious as regards visual acuity. Vision is much reduced, either by the presence of a corneal scar, by changes incident to iridocyclitis (posterior synechiæ, etc.),

or from atrophy of the optic nerve. The disease is of long duration, often lasting for months.

PATHOLOGY.—The lesions in herpes zoster ophthalmicus are found in the Gasserian ganglion and in the branches of the fifth nerve supplying the affected area. The first satisfactory report on the pathologic changes in this disease was given by Sattler in 1875, who found inflammatory lesions, with hemorrhages destroying the nerve-cells and fibres of the Gasserian ganglion. The ophthalmic division of the nerve was degenerated. The correctness of Sattler's findings has been confirmed of late by Head and Campbell, of London.

DIAGNOSIS.—Herpes zoster ophthalmicus in former years was often mistaken for erysipelas: a mistake which should not occur. The former

Fig. 140.—Herpes zoster ophthalmicus. (FUCHS.)

Appearance five days after the beginning of the disease. The vesicles follow the course of the frontal and nasal branches of the ophthalmic division of the fifth nerve.

disease is always limited to one side, never crossing the median line of the forehead or nose. There is more pain and less constitutional disturbance in herpes than in erysipelas. The vesicles of herpes are smaller, more numerous, and more circumscribed than the bullæ of erysipelas.

TREATMENT.—The treatment of this disease must be symptomatic. Elderly persons will require ferruginous preparations, tonic doses of quinin, and occasionally stimulants. Morphin or holocain may be needed to relieve the pain. Other measures for the relief of pain are the internal use of phosphid of zinc (gr. $^1/_3$ three times a day) and the application of the galvanic current. The positive pole should be placed at the vertex while the negative is applied to the affected area. The pupil is to be dilated with atropin. If tension becomes increased, as sometimes occurs in the height

of the disease, paracentesis of the cornea should be done. The skin lesions are best treated with drying powders or soothing ointments. Of the latter, the following is recommended:—

℞ Cocain ..... gr. x.
Bismuth subnitrate ..... 3iss.
Simple cerate ..... 3j.
Mix and apply locally.

The following internal remedies may be used in children and adults, the doses for children being modified according to age. For adults: —

℞ Acidi arsenosi ..... gr. j.
Pulvis piperis nigri ..... 3ij.
Extracti gentianæ ..... q. s.
M. Fiat capsulæ No. xxx.
Sig.: One capsule after each meal.

Pemphigus. — Bullous affections of the eyelids, such as dermatitis herpetiformis and pemphigus, are occasionally seen. In the former the lesions are small and grouped; in the latter they are large and scattered. Being diseases subject to frequent relapses, care must be exercised in formulating a prognosis. The treatment of these conditions is outside the domain of ophthalmology. Essential shrinking of the conjunctiva sometimes accompanies pemphigus. Contagious impetigo and various septic conditions may occasion acute processes attended with the formation of bullæ about the lids.

Lichen Ruber, Lichen Planus, and Lichen Scrofulosus occur so rarely on the eyelids and are so difficult to recognize, as well as to treat, that they should be referred to a dermatologist.

Dermatitis, if it involves the face, will also manifest itself upon the eyelids. The symptoms presented are those of acute inflammation. The most common form is that of dermatitis venenata, or poisoning, which brings about a severe chemosis and a marked conjunctivitis. In *rhus poisoning,* due to contact with "poison-ivy" or "poison-oak," the skin is of a deep-red color and the subcutaneous tissue is edematous. Patches of vesicles are present which furnish a yellowish fluid. On drying, this forms a crust. Burning and itching are distressing symptoms. The disease is self-limited. As soon as discovered the affected area should be thoroughly washed with soap and water. When the skin is unbroken, applications of dilute lead-water will give relief. Locally we may apply the fluid extract of grindelia robusta, diluted 1 part to 30 of water. A valuable application consists of equal parts of tincture of sanguinaria and water.

Dermatitis Medicamentosa is often seen to manifest itself on the eyelids. Belladonna, quinin, the turpentines, copaiba, antipyrin, arsenic, iodid of potassium, iodoform, mercury, and other internal remedies may produce this inflammation, which is best allayed by soothing applications and the discontinuance of the causative drug.

Blastomycetic Dermatitis, a rare, chronic, local infective process, beginning as a papule or papulo-pustule, often involves the lids. "The lesion slowly enlarges peripherally in the form of an indolent, flat, wart-like, or crusted papule" (Montgomery). The surface shows irregular papilliform elevations between which pus oozes on pressure. The border of the lesion is of a purple or reddish color, and is studded with minute abscesses, which can be easily recognized with a magnifying glass. The disease may remain indolent for months or years, with occasional exacerbations. The lesions may heal centrally while extending peripherally. The disease, when involving the eyelid, causes ectropion. Blastomycetic dermatitis is to be differentiated from syphilis, carcinoma, lupus vulgaris, and verrucous tuberculosis. The characteristic features are the miliary abscesses and the presence of distinct budding organisms seen on microscopic examination. The treatment includes excision of the diseased areas, which has been success-

Fig. 141.—Blastomycetic dermatitis involving the eyelids. (WALKER.)

fully practiced in several cases, the internal use of large doses of potassium iodid, and exposure to the x-rays. Cleansing or antiseptic washes or dry dressings can be used locally with benefit.

Perifolliculitis (Hordeolum; Stye).—This is an inflammation of one of the glands of the follicles of the eyelashes (Zeiss's glands). The term internal hordeolum is applied to an inflammation of a Meibomian gland. These affections are easily recognized as circumscribed inflammatory areas. They are produced by two factors: hyperemia and infection. Hyperemia may be due to one or more of various causes, including eyestrain from uncorrected or improperly corrected errors of refraction, catarrhal conjunctivitis, nasal polypi, hypertrophic rhinitis, alcoholism, etc. Infection is frequently transferred from a seborrheic eczema of the scalp by the hands. This fact has hitherto been overlooked by most ophthalmologists. Since itching exists, the patient's efforts to relieve this symptom will often

cause the spread of the disease to adjacent follicles. The crusts, resulting from desiccation of the pus, easily convey infection. Hence immediate treatment is indicated. The disease does not occur upon the eyelid proper, because no hairs exist upon it.

TREATMENT.—In the early stages the application of very cold or of very hot packs may abort the process. If this does not succeed, pus will form. If the horny epithelial layer be carefully split open, the deposit of pus in the form of a small sphere can be squeezed out and no more hordeola will occur. The incision should be made in a direction parallel with the course of the fibres of the orbicularis muscle. Antiseptic washes can be applied to the inflamed area. Internal hordeola are to be opened and evacuated from the conjunctival surface. In the type due to infection from seborrheic eczema, the patient should receive treatment from a competent dermatologist. Otherwise reinfection will occur repeatedly.

Congenital Hyperkeratosis.—In congenital hyperkeratosis of the skin, usually confounded with ichthyosis, the skin of the face and eyelids may be involved. Subjects of mild grades of the disease, who survive, sometimes show ectropion, loss of eyelashes and eyebrows, conjunctivitis and keratitis, symblepharon, and atrophy of the conjunctiva. The treatment should include alkaline and bran baths to loosen the scales, and the application of unctuous substances, of which lanolin is one of the best. The internal use of thyroid extract has been advised.

Chloasma is a pigmentary hypertrophy frequently found as a result of pregnancy and uterine diseases. In many instances the dark pigmentation is found upon the eyelids and in the neighborhood of the eyes. The treatment is difficult. Care must be exercised in the application of whatever remedy is employed, since all must necessarily be more or less irritating. Strong bichlorid solutions are probably the best.

Discoloration of the Eyelids is occasionally seen as a result of drug ingestion or accident. Thus, *argyria,* seen but seldom now, is due to the internal use of nitrate of silver. *Siderosis,* which shows itself as small brown spots, is due to the penetration of the skin by small pieces of steel. Accidental *tattooing* of the eyelids, chiefly by grains of powder, is a comparatively common occurrence. In recent cases of powder-burn the best results are obtained by placing the patient under a general anesthetic and using a nail-brush under aseptic precautions. If seen after the carbonized particles have stained the integument, puncture with the electrolytic needle will give satisfactory results, although the treatment is tedious.

Dermoid Tumors are rarely found in the eyelids. A favorite site is the region of the external angular process of the frontal bone. Rarely is the tumor found in the eyebrow. Less frequent are dermoids located at the inner angle of the upper lid. In this location the growth may have a pedicle connected with the dura mater, resembling a meningocele, in that the brain pulsation is communicated to it. Sutton states that dermoids found in the upper eyelid, unconnected with bone or periosteum, probably

arise in the fissure between the fronto-nasal plate and the fold of skin from which the lid is formed. The treatment of dermoids is excision.

Verruca, or wart, is occasionally seen upon the eyelids. The most common form in this locality is the so-called filiform variety, which is chiefly found in old persons. It is always advisable to cause the disappearance of these growths by acids, caustics, or surgical means, the best of which latter is electrolysis.

Cornu Cutaneum, or cutaneous horn, of the eyelids is comparatively small and generally involves the lower lid. It occurs in middle life, and its extirpation, with cauterization of the base, is to be advised. Epithelioma is said to be likely to follow if its spontaneous fall is permitted to occur.

Lipoma.—Fatty tumors are rare in the eyelids. They are circumscribed, soft, elastic, and lobulated. They should be excised.

Fig. 142.—Dermoid tumor. (AUTHOR.)

Hernia of the Orbital Fatty Tissue.—The fatty tissue of the orbit is normally held in place by the tarso-orbital fascia, orbicularis muscle, and skin. As a result of atrophy in elderly persons, or from trauma at any period of life, these tissues may become weakened and permit of protrusion of the orbital fat between the orbicularis muscle and the skin. The hernia can be pushed backward into the orbit. If of sufficient size to cause deformity, the protruding tissue may be readily removed through an incision made parallel with the orbicularis fibres.

Edema of the Lids is a common condition. It is often found following trauma, and is a frequent accompaniment of inflammation of the conjunctiva or infection after operations on the globe. It is found in connection with diseases of the orbit. It may follow probing of the lacrimonasal duct or the injection of fluids into the adjacent tissues. It occurs in renal and cardiac diseases, arsenical poisoning, malaria, etc. In these

conditions the edema is more marked when the patient is recumbent. In such diseases the swelling is pale and translucent. The edema due to inflammatory conditions is reddish, opaque, tense, and shining. Being merely a symptom, it does not usually call for treatment. When so extensive as to prevent opening of the eye, it can be relieved by puncture and the use of a compress bandage.

Solid Edema of the Lids.—This term is applied to a condition in which there is enormous enlargement of the eyelids, particularly of the lower lid.

Fig. 143.—Verruca of the eyelids. (VON AMMON.)

The swelling, which is often so great as to conceal the palpebral fissure, is soft and elastic, pits on pressure, is of a dusky reddish-brown color, without evidences of inflammation and without involvement of other parts of the body. In the majority of cases the disease has followed attacks of erysipelas. Some of the cases have terminated in tuberculosis of the conjunctiva. The nature of the disease is obscure. It is supposed to be a

Fig. 144.—Cornu cutaneum. (SCHAW.)

recurrent lymphangitis. Various methods of treatment—nitrate of silver, collodion, pressure, fomentations, multiple punctures, and excision—have been tried without permanent benefit.

Elephantiasis Arabum.—This rare disease sometimes involves the eyelids. It may be divided into two classes: (1) the tropical endemic type, which is due to filaria sanguinis hominis; and (2) the sporadic type, which is attributed to a variety of causes, all of which produce blocking of the lymph-channels. Thus, it follows oft-recurring attacks of ery-

sipelas. In the case reported by Walzberg (Fig. 146) hypertrophy of the left upper eyelid was present at birth. Attacks of inflammation preceded increased growth. The eyeball was displaced, the eye was amaurotic, and the cornea was hazy. The mass was successfully operated upon by Koenig. The pathologic changes in elephantiasis of the lids are an hypertrophy of the skin, particularly of the subcutaneous connective tissue, and dilation of the lymph-spaces, which are blocked, thus leading to lymphatic edema. The treatment consists in massage and surgical measures.

Lentigo (Freckles).—Small circumscribed spots or splotches of pigment occur on the eyelids as well as on other exposed portions of the skin.

Fig. 145.—Solid edema of the eyelids. (CRITCHETT.)

They usually appear about the second decade of life, but may be congenital. They do not call for treatment.

Varicose Veins.—Dilated and tortuous veins are occasionally seen in the lids, particularly in the upper lid (Fig. 147).

Nævus Pigmentosus, or pigmentary mole, is a congenital growth occurring either singly or in numbers. The form is oval or circular. The size varies from a pinhead to large tumor-like masses. The upper eyelid is more often involved than the lower. There are smooth, warty, fatty, and hairy moles. The treatment is surgical. For the larger ones excision is indicated, and, for the smaller, electrolysis is the best method.

Vitiligo and **Albinism,** the one acquired and the other congenital deficiency of the pigment of the skin, are similar in appearance, presenting a milky-white color of the integument. In the latter there is absence of

pigment in the chorioid, and nystagmus is often observed. There is no efficient treatment for either condition.

Xeroderma Pigmentosum (Kaposi's Disease). — This rare disease, of which about one hundred examples have been recorded, in most cases presents noteworthy ocular symptoms. The affection begins in early infancy with the advent of minute freckle-like spots of a brown or black color,

Fig. 146.—Elephantiasis of the upper eyelid. (After WALZBERG.)

elevated, flat or nodular, which develop under the influence of light. Its first appearance is upon the face and hands. Between the freckle-like spots are areas of depigmented skin, forming white islands. In the next stage large telangiectases appear, and finally the skin becomes atrophic, smooth, and degenerated. The skin of the eyelids is involved, and blepharitis, conjunctivitis, pigment-spots, and telangiectases of the conjunctiva and

Fig. 147.—Varicose vein of the upper eyelid. (AUTHOR.)

(Original drawing by Miss AUGUSTA BIERMAN.)

ulcers of the cornea are often present. The stage of atrophy is followed by the appearance of multiple wart-like elevations, which undergo carcinomatous or sarcomatous degeneration. These growths arise more frequently from the eyelids than from the eyeball. They ulcerate, forming fungous masses. Early in the history of the case the eyelashes fall out. The etiology of xeroderma pigmentosum is unknown. The prognosis is

unfavorable, most of the patients dying early of multiple carcinoma. Treatment so far has been without value. Panas and Monthus advise excision of the neoplasms, with cauterization of their bases.

Keratosis Follicularis (Darier's Disease).—This rare disease begins on the face or trunk, and involves the lids. It appears as a papule the size of a lentil or pea, of a dirty-red color, having a brown, black, or gray horny crust, which plugs a sebaceous duct. It is a rebellious affection, and should be referred to a dermatologist for treatment.

Molluscum Epitheliale, also known as molluscum contagiosum, is unusual, but is easily recognized. The lesions present the appearance of warts and are rather translucent, the largest being umbilicated. The disease is generally considered to be contagious and autotransferable. It is seen most frequently in children, and the eyelids are frequently attacked. The treatment is simple, consisting in enucleation of the sac and its contents. Electrolysis may be employed or an application of acid-nitrate-of-mercury ointment can be made.

Xanthoma derives its name from the yellow color which its lesions present. It is found in two forms: the plane and the tubercular. The former occurs as oval or crescentic macules varying in size from a pinhead to the thumbnail. The color is that of straw or sulphur yellow. The lesions present the appearance of a piece of chamois set into the skin. The macules occasionally become confluent, and are most frequently seen on the upper eyelid near the inner canthus. The tubercular form is of rare occurrence in this situation. Cholesterin crystals occur in abundance, there being also a marked number of new cells known as xanthoma bodies. The condition is accompanied by a fatty degeneration which, according to Pollitzer, in this situation affects the fibres of the orbicularis palpebrarum muscle. The treatment of this condition is generally unsatisfactory. Electrolysis has given good results in a few instances. The x-ray has recently been recommended.

Lupus Erythematosus is occasionally seen to involve the eyelids by extension of the process from the cheeks. It is difficult to diagnosticate and to treat, and it rarely occurs that the ophthalmologist is called upon to treat it. A large proportion of dermatologists look upon it as a form of tuberculosis of the skin.

Tuberculosis.—True tuberculosis of the skin is a disease of great rarity. It is found in the form of discrete, shallow, painless ulcers with eroded, irregular edges. When the crusts are removed a reddish-yellow granular surface is exposed. The ulcers never heal. They spread continuously, and coalesce with other ulcers to form large areas. Crocker states that the diagnosis is difficult in the absence of signs of general tuberculosis. The disease is found in regions where the skin and mucous membrane join. Since tubercle bacilli are found in lupus vulgaris, scrofuloderma, tuberculous ulceration, and tuberculosis verrucosa cutis, the term tuberculosis of the skin is loosely applied to affections which, however similar they may be

microscopically and pathologically, present marked clinical differences (Crocker). Tuberculosis of the skin offers an unfavorable prognosis.

Lupus Vulgaris, which is common on the European continent, is rare in this country. When present upon the face or nose it may extend to the eyelids. It is characterized by the presence of papules, nodules, and patches, which either ulcerate or atrophy, leaving scars. It almost invariably appears before puberty, and is more frequent in females than in males. It begins as pinhead spots of a reddish color which change into nodules. These present a brownish, translucent appearance, likened by Hutchinson to apple-jelly. After a time, which may be months or years, the nodules coalesce, become elastic to the touch, and ulceration occurs, which spreads and cicatrizes spontaneously. When the eyelids are attacked there may be cicatricial ectropion, or the lids may be entirely destroyed. In the latter case the eyeball will be attacked. The disease, which is of long duration, may remain unrecognized until seen by a dermatologist, since the diagnosis is difficult. The treatment is complete removal of the diseased tissue.

Syphilis.—Syphilis of the eyelid may be present as primary, secondary, or tertiary lesions, or as hereditary manifestations. *Chancre* (Fig. 7, Plate VII) presents as an ulcer with indurated base. Until the development of secondary symptoms the diagnosis may be in doubt. The conditions with which it is most likely to be confounded are the pustules of vaccinia and tuberculous ulcer. In chancre the pre-auricular gland is always, and the submaxillary is frequently, indurated. Only a careless or incompetent observer could mistake a chancre for a stye, chalazion, or dacryocystitis. Chancre may appear at any period of life. It is more often found on the lower than upper lid, and in men more frequently than in women. The infection may be carried by unclean fingers, towels, instruments, by kissing, or by the practice, common among Russians, of removing foreign bodies by licking. Tepljaschin, in a small town in Russia, met with thirty-four cases of chancre of the eyelids caused by a female quack who treated granular conjunctivitis by everting the lids and licking them. Not infrequently physicians, while treating the throats of syphilitics, have become inoculated by the patient's saliva projected by coughing. In the treatment use may be made of the yellow wash locally and mercury internally or by inunction. The prognosis is favorable.

Serpiginous Syphilide of the Eyelid may closely resemble lupus or tuberculosis, and produces extensive destruction unless checked by appropriate internal treatment.

Gumma is probably the most frequent of the syphilitic diseases of the lids. The lid becomes swollen and tense. Ulceration follows, the ulcer having an irregular, eroded, "punched-out" appearance. Its floor is covered with dirty-yellowish or gray *débris,* and if unchecked there may be extensive destruction of the tissues. In the absence of a history of infection the diagnosis may be difficult. The syphilitic ulcer presents an infiltrated base, but not the parchment-like indurated base of chancre (de Beck).

Rodent ulcer is found in older persons, as a rule. Tubercular ulcers of the eyelid have their origin in tuberculosis of the conjunctiva. They are exceedingly rare. There is a profuse purulent discharge and early involvement of the cornea. A suppurating chalazion or hordeolum will give a different history and will run a different course. The prognosis of gumma of the lid is favorable, the condition yielding to iodid of potassium internally and mercurial salve locally. Cauterization is contra-indicated.

Leprosy is a general affection of the human organism and one not easily recognized by the non-expert. So far as the eyelids are concerned, its local forms are thickening of the lids, the presence of tubercles upon them, or ulceration of the tissues. The eyebrows and eyelashes are apt to

Fig. 148.—Gummata of the eyelids. (KEBER.)

turn white or they may drop out. In advanced cases of anesthetic leprosy, when mutilations occur, there is observed an ulcerated process involving the eyelids and even the ocular globe. A distinguishing symptom between this and lupus is the total absence of pain in the former. All treatment for leprosy has proven ineffectual, although chaulmoogra-oil is said to be useful. Recently the hypodermic injection of Calmette's antivenene serum has given promise of good results (Dyer).

Keloid is a connective-tissue neoplasm which rarely attacks the eyelids. It is frequently the result of a traumatism, and is often seen in the negro or one who has in him negro blood. The treatment is unsatisfactory. Electrolysis or the x-ray may be tried.

Plexiform Neuroma of the Eyelid (Neurofibroma) is a rare growth which is really a fibroma developing from the sheaths of peripheral nerves, there being no new development of nerve-fibres. The growth, which shows a peculiar predilection for the upper eyelid, is either congenital or appears in early infancy. The lid becomes greatly enlarged and of elephantiasis-like appearance, and ptosis results. The tumor is soft in general, with localized cord-like spots which can be traced backward into the orbit. In some of the recorded cases the face and eyeball have been involved. Usually the growth is not painful. The adjacent skin presents numerous spots of brownish-yellow color. Microscopically such growths are composed of nerve-bundles imbedded in masses of connective tissue. The treatment is excision. If incompletely removed the growth will return.

Fibroma of the Eyelid.—This disease, known also as the painful subcutaneous tubercle of Wood, may be found in the lid as a small, hard, rounded mass, usually remaining stationary after reaching a certain size. It is subject to attacks of radiary pain. Such a growth is usually freely movable, and its true nature is likely to be revealed only after removal. Microscopic section shows the mass to consist of bundles of densely packed fibrous tissue containing numerous blood-vessels.

Nævus Vasculosus is frequently seen and is easily recognized. It is congenital and occurs about the eyelids not infrequently. Nævi are roundish or irregular in shape and size, and bright red, violaceous, or blue in color. They are very vascular, being essentially composed of dilated or hypertrophied blood-vessels. Angioma cavernosum is very vascular and at times pulsating; angioma simplex is non-elevated. The most common variety involving the integument of the eyelids and the sclera is the nævus flammeus, or port-wine mark. Vascular tumors of the lids may bleed either spontaneously or in response to slight trauma. The best treatment of nævus vasculosus is by means of electrolysis, and even then it is prone to recurrence.

If the case comes under treatment shortly after birth, painting with collodion may give good results.

Lymphoma of the Eyelids (Lymphangioma).—This form of growth, which is rarely observed, appears as an enlargement of the eyelid, either alone or associated with similar growths in the orbit, the mouth, axilla, etc. In the eyelid a lymphoma forms an elongated sausage-shaped tumor, elastic and painful. The skin over the mass is often tense, shining, and traversed by dilated veins. The surface of the growth is slightly lobulated, and may be small or so large as to conceal the eyeball. Lymphoma develops in the course of leukemia and pseudoleukemia. Optic neuritis, retinitis, and retinal hemorrhages are often present in these diseases. Lymphoma of the eyelids may appear at any period of life, but is most frequent in middle age. The prognosis is unfavorable. Extirpation of the growths seems to be of only temporary value. Bronner has recorded a case which was cured by the prolonged internal use of arsenic.

Carcinoma of the Lid (Epithelioma; Rodent Ulcer).—Cancer of the lid is a malignant growth consisting chiefly of epithelial cells and arising from mucous or cutaneous surfaces. On microscopic section it presents characteristic cell-formations called "nests." While epithelioma and rodent ulcer present practically the same microscopic changes, their clinical features are sufficiently different to demand separate descriptions.

Epithelioma develops most frequently from mucocutaneous junctions. In its earliest stage it may appear as a wart, a nodule, or a fissure. After a time ulceration occurs, the ulcer having indurated, everted, or undermined edges. Sooner or later the neighboring lymphatic (pre-auricular,

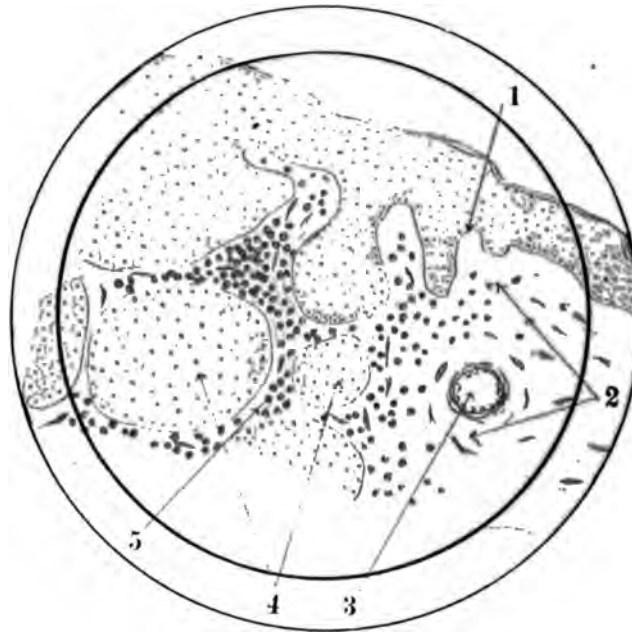


Fig. 149.—Microscopic section of an epithelioma of the eyelid. (AUTHOR.)

(Original drawing by DR. CARL FISCH.)

1, Boundary between normal and epitheliomatous tissue. *2*, Connective tissue. *3*, Small vessel. *4*, Epithelial nests. *5*, Small round-cell infiltration.

submaxillary) glands are involved. The process extends and ends in death by hemorrhage or exhaustion. There is little pain, of an itching or shooting character, in the earlier stages of the disease. Later, when the destruction of tissue is extensive, the suffering is severe. The disease occurs chiefly in persons past the middle period of life, and is more frequent in men than in women.

DIAGNOSIS.—Epithelioma is to be differentiated from syphilitic nodules and gummata, rodent ulcer, lupus, chancre, and tuberculosis. In syphilitic processes the progress of the disease is usually rapid and the history is that of infection. In gummatous ulceration there is no hardness around the ulcers, which are multiple and punched-out and present an abundant puru-

lent discharge. In epithelioma the process is slow and the discharge scanty, thick, and bloody. Chancre may be differentiated by its history and rapid development. When there is any doubt as to the diagnosis, energetic antisyphilitic treatment should be tried for a few weeks. Lupus presents multiple lesions, which begin in childhood or youth and usually are associated with lupus of the nose or cheek. Lupus shows an ulcer of a saucer shape, with an abundant, non-purulent discharge, free from blood, and not offensive. Adjoining the ulcer are numerous soft, brownish, semitranslucent tubercles. Accompanying the process is considerable inflammation, causing the lid to be red and swollen. The disease often begins in the palpebral conjunctiva and eats into the lid. It is possible, however, for epithelioma to be ingrafted upon lupus. Rodent ulcer is described in a separate paragraph. A broad, flat, slowly growing epithelioma which has destroyed the lid-margin and invaded the conjunctiva, which presents a mammillated appearance, may so closely resemble tuberculosis that only a microscopic examination can determine the diagnosis.

PROGNOSIS AND TREATMENT.—Left to itself, epithelioma is fatal. Removed early, it offers a fair prognosis. If neglected until after glandular involvement has occurred, the prognosis is unfavorable, return of the growth after excision being the rule. Treatment by the Roentgen ray and by Finsen's light is too recent to enable judgment to be passed as to its value. If neglected, epithelioma of the eyelid will extend to the eyeball and necessitate enucleation. In the treatment escharotics are not to be ranked with excision. At the time of removal of the growth by means of the knife a suitable plastic operation should be made to cover the defect.

Rodent Ulcer (Jacob's Ulcer; Cancroid Ulcer). — This is a slowly growing ulcer of the face, generally involving the eyelids. Histologically it is a carcinoma, and is often described as a form of epithelioma. Clinically it presents features which serve to distinguish it from epithelioma. The essential facts of rodent ulcer were described by A. Jacob, in 1827, in these words: "The characteristic features of this disease are the extraordinary slowness of its progress; the peculiar condition of the edges and surface of the ulcer; the comparatively inconsiderable suffering produced by it; its incurable nature, unless by extirpation; and its not contaminating the neighboring lymphatic glands." Jacob's ulcer begins as a small nodule with depressed centre, of firm consistence and brownish-red color. After a variable period the skin breaks, leaving an ulcer with undermined edges and infiltrated border. This becomes broader and deeper, destroying all tissues, including the bones. It extends in the direction of the orbit, and often the eyeball falls out. Its etiology is obscure and the prognosis must be guarded. The treatment is excision. Recently the treatment by the Roentgen ray and Finsen's light has given encouraging results.

Sarcoma of the Eyelid.—About fifty cases of sarcoma of the eyelid have been published (Veasey). The youngest patient was seven months of age and the oldest seventy-six years. The size varied from that of a pin-

head to that of an apple. In rare instances all four lids have been involved, and in 16 per cent., principally children, the disease caused death. There was more or less pigment in 26 per cent. of the cases. The disease returns in probably 50 per cent. In four cases the neoplasm was attributed to trauma. The symptoms are practically those of chalazion. A tumor is present between the skin and tarsus. It develops gradually without pain.

Fig. 150.—Rodent ulcer. (GRINDON.)

In the pigmented cases the discoloration will aid in the diagnosis, which otherwise must remain in doubt until after microscopic examination. The growth is encapsulated to a greater or less extent in about 14 per cent. of cases. The prognosis must be guarded. The treatment is excision. The growth may return locally or metastatically. Veasey states that 38.5 per cent. were spindle-cell, 38.5 per cent. round-cell, and 23 per cent. mixed-cell sarcomas.

Adenoma of the Meibomian Glands is a rare form of tumor. The growth involves the lids, which become nodular. Eversion of the lid shows yellowish, nodular masses involving the Meibomian glands. The growth causes the eyelid to become stiff and board-like. Knapp has reported a case and has found nine others in literature. The mass should be excised and examined microscopically.

Noma of the Eyelids (Spontaneous Gangrene; Phagedenic Ulceration).—This rare condition has been observed by Rushmore, Derby, Hilbert, Marlow, Roger and Weill, and Morax. The disease appears in emaciated infants and children. It is characterized by swelling of the lid, the formation of a pimple which is soon converted into a pustule and is followed by ulceration, and the presence of a thin conjunctival discharge. The skin, conjunctiva, and intervening structures soon break down, and may be partly or entirely destroyed. The ulcer presents sharply-defined, undermined edges, bordered by a zone of darkly-congested skin. There is greenish pus and a dirty slough. The cornea is opaque and ulcerated and perforation results. The ulceration may spread into the eyebrow and on to the cheek. The bacterial cause of noma has not been determined. The prognosis in these cases will depend on the time when the patient is brought for treatment and on the general condition. Cleansing and antiseptic treatment will be in order locally, and supportive measures internally. In Marlow's case Labarraque's solution was used with benefit. The administration of diphtheria antitoxin has been strongly recommended.

Colloid Degeneration of the Skin, a rare disease of which five authentic cases are on record, involves at times the forehead, bridge of the nose, eyelids, and, in some instances, the conjunctiva. The diagnosis can be made only by a competent dermatologist.

Hyperidrosis, or excessive sweating, is noted in connection with the disease occurring on the face and body. It may be confined to the lids of one eye when there is unilateral facial hyperidrosis. In this case it indicates an irritation or lesion of the sympathetic nerve.

Chromidrosis, or colored sweat, is occasionally observed affecting the eyelids, and may be either yellow, red, or blue. It may be that only one-half of the lids is affected, and when this occurs it is the outer half. The majority of these patients are women. In hysterical subjects the disease is often simulated, ordinary coloring substances—such as indigo, soot, and plumbago—being used to carry out the deception.

Phosphoridrosis and **Uridrosis,** or phosphorescent and urinous sweat, are seen about the eyelids and always in connection with a generalized form of the diseases, which depend upon a general involvement of the system.

Sudamina, which consists of pinpoint- to pinhead- sized vesicles, usually occurring upon the hands, is also rarely observed upon the eyelids. It is caused by a too rapid formation of sweat, and is observed in summer. The application twice a day of a 1-per-cent. strength solution of chromic acid will rapidly cause its disappearance.

The disorders of secretion of the cutaneous glands of the skin, which are most frequently seen, embrace those of the sebaceous and of the sweat-glands.

Seborrhea is observed at times upon the upper eyelid in the dry form and on both lids in the oily form. Here it is not only necessary to use internal treatment, but local applications, preferably in the form of sulphur ointment or a mixture of equal parts of 5-per-cent. oleate of mercury and precipitated sulphur, mixed with a base of cold cream (unguentum aquæ rosæ).

Miliaria, or lichen tropicus, better known as "prickly heat," is a common affection of the eyelids in summer. It is easily recognized, and occurs most frequently in fat babies. In these a mild conjunctivitis is a very common accompaniment. An efficient as well as rapid treatment of the skin eruption is by the application, three or four times daily, of a mild solution of sulphate of copper (gr. x to ℥vj of water).

Milium is perhaps at times a retention cyst of the sebaceous glands. Recent investigation has shown that the small tumor is an agglomeration of the prickle layer of the epidermis which has become snared off into the corium during embryonic life. It is often observed, chiefly about the lower eyelid, but often on the upper one also. While occasionally congenital, it is usually not observed until after puberty. Milia may occur sparsely or may be thickly studded over the affected area. The lower lid is more often affected than the upper one, and the tendency of the distribution of the lesions is said to be rather toward the outer canthus. When occurring in patches, the disease may resemble xanthoma. The treatment is simple, consisting in the enucleation of every milium with a milium needle, at the same time destroying the lining membrane of the little cyst to prevent the condition from recurring.

Acne Rosacea occurs about the eyelids in severe cases and is inclined to be chronic in this locality, as the lids are not only congested, but involved in a certain degree of chemosis. The diagnosis is rendered easy by the presence of the trouble in the face. Lotions are to be avoided, as they are apt to produce irritation of the conjunctiva. Ointments containing from 15 to 30 grains of sulphur to the ounce of simple cerate, applied twice daily, will act favorably.

Asteatosis, or the lack of secretion of sebum, requires particular attention, as its pronounced stage will produce ectropion. Here the regular application of animal fats and the internal administration of arsenic will procure the best results. Little hope of permanent recovery can be held out.

Steatoma, which is supposed to belong to the class of dermoids, is not infrequently seen about the lids. As a rule, there is but one, and most usually it is about the outer canthus of the upper or lower lid. The growth of these cysts is slow, and they are due to some injury to the opening of the sebaceous gland. The tumor is round and smooth and varies from the size of a pinhead to that of a hazelnut. It contains broken-down epithelial

PLATE VII.

External Diseases of the Eye.

Fig. 1.
Distichiasis and Trichiasis.

Fig. 2.
Chalazion.

Fig. 3.
Blepharitis Marginalis.

Fig. 4.
Symblepharon.

Fig. 6.
Ectropion.

Fig. 5.
Entropion.

Fig. 7.
Chancre of the Conjunctiva.
(De Schweinitz.)

Fig. 8.
Epithelioma of the Corneoscleral
Region. (Oliver.)

External Diseases of the Eye.

The diseases of the glands of the eyelids, which are most frequently seen, embrace those of the sebaceous and of the sudoriferous glands.

Seborrhœa is observed as three varieties upon the upper eyelid in the dry form and on both lids in the oily form. Here it is not only necessary to use internal treatment, but local applications, preferably in the form of sulphur ointment (_____ to the drachm of cosmoline) or of coal tar preparations (_____ and precipitated sulphur, mixed with a lotion of mild mercurial sublimate).

Miliaria, or lichen tropicus, better known as "prickly heat," is a common affection of the eyelids in summer. It is easily recognized, and occurs most frequently in fat babies. In these a mild conjunctivitis is a very common accompaniment. An ointment as well as rapid treatment of the skin eruption is by the application, three or four times daily, of a mild solution of sulphate of copper (gr. x to ℥ij of water).

Milium is perhaps as often a retention cyst of the sebaceous glands. Recent investigations have shown that the small tumor-like accumulations of the products of the sebaceous glands which has become shriveled up into the cystic sac during embryonic life. It is often observed, closely about the lower eyelid, but often on the upper one also. While occasionally congenital, it is usually not observed until after puberty. Milia may occur sparsely or in large numbers scattered over the affected area. The lower lid is more often affected than the upper one, and the tendency of the distribution of the tumors is said to be rather toward the outer canthus. When occurring in patches, the lesions have resemble xanthoma. The treatment is simple, consisting in the evacuation of every cystigen with a needle, at the same time destroying the lining membrane of the cyst, and to prevent the recurrence of the tumor.

Blepharitis Marginalis. Fig. 3.

Syringobpharon. Fig. 4.

Xanthelasma. Fig. 5.

Epithelioma of the Conjunctiva. (Oliver.) Fig. 6.

Chancre of the Conjunctiva. (De Schweinitz.) Fig. 7.

Entropion. Fig. 8.

Blepharitis Ciliaris is observed about the eyelids in acute cases and is marked by an increase in the lymph, as the lids are not only congested, but indurated to a certain degree of chemosis. The diagnosis is rendered easy by the presence of the trouble in the lash. Lotions are to be avoided, as they are apt to produce irritation of the conjunctiva. Ointments containing from 15 to 30 grains of sulphur to the ounce of simple cerate, applied twice daily, will act favorably.

Anomalies of the lash of eyelashes, require particular attention, as the pronounced stage will produce entropion. Here the regular application of actual fire and the internal administration of arsenic will produce the best results. Little hope of permanent recovery can be held out, however, when the disease is allowed to belong for years. Trichiasis, or misdirection of the lashes about the lids. As a rule, there is but one, and most usually it is about the outer canthus of the upper or lower lid. The growth of these cases is slow, and they are due to some injury to the opening of the sebaceous gland. The tumor is round and smooth and varies from the size of a pinhead to that of a hazelnut. It contains broken-down epithelial

PLATE 7

cells, forming a pultaceous mass. These tumors have well-defined walls. They occur in children as well as in adults. The treatment is to incise the tumor, empty it of its contents, and tear out the lining sac. A rapid recovery follows, and a well-done operation in a few days shows no sign of having been performed.

Chalazion (Meibomian Cyst; Tarsal Tumor; Hailstone) is a tumor of the eyelid which is generally described as a Meibomian cyst. It is not directly a cyst, but a mass of granulation tissue scantily supplied with vessels and tending to necrosis. The cyst develops only secondarily by the breaking down of granulation tissue. The tumor starts primarily, not from glandular tissue, but from infection, and is due frequently to the tubercle bacillus. Histologically a chalazion is composed of typical tuberculous tis-

Fig. 151.—Microscopic section of a chalazion. (AUTHOR.)

(Original drawing by DR. CARL FISCH.)

1, Meibomian gland cut across. *2*, *2*, Areas of round-cell infiltration. *3*, Artery. *4*, Conjunctival epithelium. *5*, Giant cell. *6*, Epithelioid cell.

sue, presenting numerous round cells, epithelioid cells, and occasionally a giant cell (Fig. 151). Recently Hála examined twelve cases of chalazion histologically and bacteriologically and constantly found a bacillus identical with the xerosis bacillus. He believes that these bacilli, which often are found normally in the conjunctival sac, are rubbed into the tissues, where they produce chalazia. Fisch, of St. Louis, has demonstrated the presence of tubercle bacilli in giant cells found in chalazia.

SYMPTOMS.—A chalazion forms a round, hard, small tumor situated commonly in the middle of the lid near its border. It is more frequent in the upper than in the lower lid, and is more often found in adults than in children. Its progress may be in the direction of the conjunctiva (internal chalazion) or toward the skin (external chalazion). When the lid

is everted the conjunctiva over the chalazion is found to be red and thickened, and in advanced cases a bluish or grayish spot is seen corresponding to the thinned tarsus. Several chalazia may be present at the same period or single tumors may develop from time to time. As a rule, chalazion is painless, and causes inconvenience only when it attains a considerable size, in which case the eyelid may droop and the tumor will cause a deformity. Not infrequently chalazia disappear spontaneously. When perforating the conjunctiva, the chalazion leaves granulations and thus may simulate a malignant growth. The growth of a chalazion, while generally slow, may be rapid. Acute chalazion may develop rapidly, with pain, swelling, and localized conjunctivitis. The growth may undergo suppuration, in which case it resembles a stye.

ETIOLOGY.—Chalazion is due to infection. It is aggravated by errors of refraction and by conjunctivitis. At least 20 per cent. of individuals with chalazion suffer with tuberculosis; in 70 per cent. conjunctivitis and blepharitis are present; in 30 per cent. there is lacrimal stricture; 27 per cent. are ametropic, and 12 per cent. have scrofula (Strzeminski).

DIAGNOSIS.—Acute chalazion is to be differentiated from hordeolum by the diffuse appearance of the latter. The "pointing" of hordeolum is noticeable. Chronic chalazia may be mistaken for sebaceous cysts. Here the firm attachment of the chalazion to the tarsus will clear the diagnosis. Sarcoma of the eyelid may so closely resemble chalazion that the diagnosis can be made at an early stage only by microscopic examination. Recurrence of a lid tumor soon after removal will cause suspicion of malignancy.

TREATMENT.—Acute chalazion should be treated by incision and the frequent application of hot compresses. Chronic chalazion may sometimes be cured by the daily use of massage and the application of mercurial or iodin ointments. The iodid of cadmium is recommended because of not staining the skin. If, after several weeks' trial, this plan of treatment fails, the case will call for surgical intervention as described elsewhere in this chapter. In cases of recurring chalazia it will be necessary to give careful attention to the general health and to the correction of errors of refraction.

Alopecia and **Alopecia Areata**, or progressive baldness and baldness in spots, are observed to affect the eyebrows. Local stimulating applications and appropriate general treatment will be found to be the most efficient means to procure good results. *Hysterical alopecia of the lids* is sometimes seen in neurotic females and in hysterical children of both sexes. Such subjects systematically pull out the cilia.

Parasitic Skin Diseases are very rarely observed upon the eyelids, that which is most ordinarily seen being due to the pediculus pubis. These pediculi are occasionally found to infest the eyelashes of infants, and in consequence to produce tinea, leading to acute conjunctivitis and blepharitis (phtheiriasis; blepharitis pediculosa). The treatment consists in picking off the pediculi and applying a soothing lotion to the affected eye.

Mosquitoes, fleas, and bedbugs occasionally attack the eyelids of sleeping

persons and produce lesions. These may be small red papules with a scarlet central point or wheals, and should be treated by means of a soothing ointment. The parasite must be looked for and removed.

Favus of the Eyelids is an exceedingly rare disease, only five cases having been reported. The disease first shows itself as yellowish-red vesicles, which are painful. Later there is a dry, fissured crust, elevated above the level of the surrounding skin. The crust is of a sulphur-yellow color, and its centre shows a depression, with a variety of colors varying from white to sepia brown. Microscopic examination shows the presence of the sporidia and mycelia of achorion Schoenleinii. The treatment consists in the use of an oiled compress in the evening and frequent washings with bichlorid solution (1 to 4000).

Cysticercus of the Eyelid is an exceedingly uncommon affection. It may involve either eyelid or may be found beneath the skin of the eyebrow.

Fig. 152.—Varioloid of the eyelids. (Von Ammon.)
The patient had suffered a previous enucleation.

It develops without inflammatory symptoms, and forms a round, elastic, movable tumor which is likely to be mistaken for an ordinary cyst. Operation will show its true character.

Variola manifests itself on the eyelids in the papular, vesicular, and pustular stages. The integument becomes swollen and edematous as well as bright red in color. A more or less marked conjunctivitis shows itself, and it is sufficiently severe to cause the escape of an appreciable quantity of muco-purulent secretion. The eyelids themselves burn and itch. Efforts to relieve this by rubbing with the fingers will produce inoculation of non-affected parts of the skin and of the conjunctiva. An efficient means of treating the itching is by the use of red rays, which not only alleviate this symptom, but prevent subsequent pitting. In any event, the further development of pus must be arrested. This is to be done by applications and instillations of efficient antiseptics. The eruption, although not commonly

severe on the lids, being usually discrete in its distribution, at times is aggregate and even confluent.

Vaccinia of the Eyelid is rarely observed. The virus is carried by the fingers to the margin of the lid, where it causes an ulcer. Pain, swelling, and redness of the lid, conjunctivitis, and tenderness and enlargement of the pre-auricular and submaxillary glands are present. There is generally some fever and malaise. The condition is readily differentiated from syphilis by the history of the case. In about a week crusts fall from the lid, the ulcer heals, and recovery is complete. Aside from cleanliness, these cases require no treatment.

Varicella is not frequently seen to manifest itself in the eyelids. When it does, it is not severe in character, perhaps only one lesion being found. The subjective symptoms are practically *nil*. There is little, if any, itching until after a crust forms. Patients will invariably make attempts to remove this, leaving a marked and comparatively deep pit. The scar is marked, but on account of the scarcity of lesions it produces only slight deformity. The treatment of the disease is purely symptomatic.

Scarlatina manifests itself about the eyes in the form of a catarrhal conjunctivitis, accompanied by a more or less severe chemosis. The eyelids are of a scarlet color, more or less marked edema exists, and an intense sense of burning is present. The edema is so severe in some cases that the patient is unable to open the eyes. It is likely to be mistaken for erysipelas by those who do not carefully observe the other symptoms present. When the stage of desquamation has been reached, large, thin scales will separate from the eyelids. Care should be taken that these are not torn off. Mild astringent ointments should be used in this stage, whereas cooling lotions are indicated in the acute stage of the disease. The conjunctivitis should be treated appropriately.

Rubeola, or measles, also attacks the eyes in a manner similar to scarlatina. In this case the eyelids present the characteristic splotches observed upon other parts of the skin. The conjunctival inflammation is distinctly catarrhal. The involvement of the lids and conjunctiva is of a mild type, and is to be controlled by the use of sedative and mild astringent lotions. When desquamation begins a neutral ointment should be applied.

Blepharochalasis, a relaxation of the palpebral skin, has been described by Fuchs, Hotz, Starkey, and others. Following recurrent angioneurotic edema, the skin loses its attachment to the tarsus, so that, when the eyelid is raised and slides over the eyeball, the skin is not retracted with it, but falls over the retracting eyelid. The condition can be cured by excising a portion of redundant skin and anchoring the palpebral skin to the tarsus.

Emphysema of the Eyelids.—A collection of air in the cellular tissue of the eyelids not infrequently follows a compound fracture of the nasal bones or an operation upon the lacrimal canal. In either case, the patient, by blowing the nose, forces air into the cellular tissue. There is pain, sudden and great swelling of the lid, and air-tumefaction. Palpation gives

a sense of crepitation and a peculiar soft feeling like that of a feather bed. The air comes from one or more of the adjacent cavities: the nasal fossa, ethmoidal cavity, frontal sinus, or antrum of Highmore. Emphysema may follow the operation of opening the ethmoid cells. Douglass has recorded cases in which emphysema of the upper lid followed an attempt to blow secretions from the nose, in patients who had suffered no operation or trauma. In these cases there was probably a pathologic opening between the ethmoid cells and the orbit. The prognosis of emphysema is favorable. A compress bandage should be applied.

Blepharitis Marginalis (Blepharitis; Ophthalmia Tarsi; Tinea Tarsi; Seborrhea of the Palpebral Margins; Blepharitis Ciliaris; Blepharitis Ulcerosa; Psorophthalmia; Sycosis Tarsi; Blepharo-adenitis). — These terms are applied to several varieties of chronic inflammation involving the margins of the eyelids (Fig. 3, Plate VII).

HYPEREMIA OF THE LID-MARGINS is present in persons who do an excessive amount of work at near points (writers, watchmakers, sewing-women) or follow their vocations in a vitiated atmosphere or under unfavorable illumination. It is common in persons with errors of refraction and in those who are addicted to the use of alcohol and tobacco. When, in addition to hyperemia, small scales form on the lid-margins, the condition is known as *blepharitis squamosa*. If the lid-border becomes covered with yellow crusts, which, when removed, leave ulcerated areas, the disease is known as *blepharitis ulcerosa* or *eczematosa*.

1. In Blepharitis Squamosa (seborrhea of the lid-border; blepharitis ciliaris) the lid-margin is reddened, the palpebral conjunctiva is hyperemic, and the space between the cilia is filled with small, thin, whitish scales resembling dandruff. The scales can be removed imperfectly by washing, or thoroughly with the forceps, leaving the lid-margin reddened and succulent, but not ulcerated. The cilia fall out easily. They soon grow again. Instead of the scaly formation mentioned, the lid-margins may present yellow crusts, with no underlying ulceration, due to the drying of excessive sebaceous secretion. Patients with blepharitis complain of a burning or itching sensation. Generally there is also present a chronic type of catarrhal conjunctivitis, with a slight formation of mucus, which may at times spread over the cornea and cause momentary blurring of vision. The eyes are sensitive to light, heat, and dust. They tire easily and styes often are present.

2. Blepharitis Ulcerosa.—This is a more severe process. The lashes are matted together with yellow crusts. On their removal a raw bleeding surface remains. From the centre of each yellow elevation a cilium arises. The lid presents numerous abscesses from suppuration in the hair-follicles and their sebaceous glands. It shows an irregular, worm-eaten condition from the cicatrices of healed abscesses and the extension of those yet existing. Where the cicatrices exist, the cilia are absent because of destruction of their follicles. When blepharitis ulcerosa has existed for a long time,

few cilia remain, and those present are often found in small tufts glued together with dried secretion. This form of lid inflammation is much more serious in its sequelæ and prolonged in its course than the squamous variety. The sequelæ are chronic catarrhal conjunctivitis, destruction of the cilia (madarosis), trichiasis, hypertrophy of the lid-margin, which becomes rounded and bordered with fleshy-looking conjunctiva (tylosis), ectropion, persistent lacrimation from eversion of the puncta, blepharospasm, photophobia, and eczema of the face. These conditions react upon the blepharitis, thus establishing a vicious circle. When, in an old case of blepharitis ulcerosa the "lid-border becomes smooth, red, glazed, everted, thickened, weeping, and destitute of lashes," the term lippitudo is applied.

Etiology and Pathology. — Blepharitis marginalis is a common disease among children and scrofulous subjects. It is particularly frequent in blondes. Seborrhea of the lid-margin may follow in the wake of a similar disease in the hairy scalp. Eczema of the lid-margins is generally associated with eczema of the scalp, ear, and face. Uncorrected or improperly corrected errors of refraction certainly increase the local trouble, if indeed they do not cause it. Often blepharitis follows one of the exanthematous diseases of childhood. It is frequently associated with naso-pharyngeal disease and with stenosis of the lacrimo-nasal duct. Staphylococci are found in the eczematous form of blepharitis. A minute parasite inhabiting the sebaceous follicles, demodex folliculorum, is regarded as a cause; and a vegetable growth, the trichophyton fungus has been observed in some cases. The etiologic importance of the demodex folliculorum is doubted by Sulzer, who found it in the normal lids of one person in six examined. Raehlmann has applied the term *blepharitis acarica* to those cases in which this parasite has been found.

From what has been stated it is evident that the pathologic changes in this disease concern chiefly the glands and cilia in the simple forms, and in addition thereto the adjacent structures are involved in the ulcerous form.

Diagnosis.—Blepharitis may be confounded with conjunctivitis. If, after removal of crusts, the skin of the lid-margin is normal, the case is one of conjunctivitis. In blepharitis this area is either reddened or reddened and ulcerated. In phtheiriasis of the lids the lid-borders look dark from the presence of the nits of crab-lice on the lashes. Careful examination under a magnifying glass will clear the diagnosis. This affection, which itself sometimes causes blepharitis, is found almost exclusively in children.

Prognosis.—Blepharitis squamosa offers a favorable prognosis. The eczematous form is somewhat rebellious to treatment. Either form may persist for months or years, yet the majority will be cured. For madarosis there is no efficient treatment.

Treatment.—Many patients with blepharitis require tonic treatment. Attention should be given to the condition of the alimentary tract and the

naso-pharynx. The proper correction of errors of refraction or of muscle imbalance is of great importance. As regards local treatment, the two main requirements are: (1) to keep the lid-margins clean and (2) to apply appropriate and soothing remedies. The first object can be attained by the daily removal of scales and dried secretion by means of forceps. This should be done by the surgeon himself. If, after thorough cleansing of the lid-margin, there remains a reddened and succulent area without ulceration, an ointment of ammoniated mercury or yellow oxid (gr. i or ij to 3j) should be rubbed into the roots of the cilia. Persistent local treatment, together with attention to the general health and the wearing of proper glasses, will give favorable results in this, the squamous form of blepharitis.

In the eczematous form of the disease removal of the dried crusts will expose areas of ulceration. There will be free bleeding and some pain connected with the thorough removal of the crusts. After cleansing with peroxid of hydrogen, each ulcer should be touched with a strong solution of silver nitrate or with the mitigated stick. The resulting scab should not be disturbed. After it has dropped off, the lid-margin is to be cleared of all *débris,* and any bleeding points remaining are treated similarly. Each day the ointment of ammoniated mercury or the yellow-oxid ointment should be applied to the lid-margins. After the ulcers have healed the surgeon should examine for errors of refraction and properly correct them.

Conjunctivitis, lacrimal stenosis, entropion, ectropion, and naso-pharyngeal disease, which may be present, should be given appropriate treatment.

Among other remedies for blepharitis are the oils of cade, rue, or juniper; creolin ointment, Hebra's compound diachylon ointment, boric-acid ointment, solution of formalin, sulphur ointment, pyrogallol salve, and solution of chloral hydrate.

OTHER DISEASES OF THE EYELIDS.

Lagophthalmos (Hare's Eye).—This term, derived from the ancient belief that the hare sleeps with open eyes, is applied to cases in which, owing to drooping of the lower eyelid, there is inability to close one or both eyes. Attempts to close the lids cause the globe to roll inward and upward, the lids remaining open. The cornea, being exposed, becomes dry; dust and foreign bodies lodge upon it; the tears, owing to paralysis of Horner's muscle, no longer pass into the tear-ducts, but flow on to the cheek; and soon a corneal ulcer forms which may end in perforation and loss of the globe. The lesion may be lagophthalmic keratitis or xerosis of the cornea (see chapter on the cornea). The condition may be attributed to: (1) paresis or paralysis of the facial nerve; (2) spasm of the upper eyelid, due to disease of the third nerve; (3) shortening of the lids; (4) extreme proptosis, due to exophthalmic goitre or the growth of orbital

tumors; (5) enlargement of the globe, as in hydrophthalmos; (6) severe and exhausting diseases in which the sensitiveness of the cornea is lost and the winking reflex is absent. Cases of lagophthalmos are usually classified as paralytic and non-paralytic.

In lagophthalmos from disease of the facial nerve, the central lesion may be cortical, intracerebral, or nuclear. In the peripheral form the lesion may involve the seventh nerve within the temporal bone, in the canal of Fallopius (intracranial), or after its escape from the stylo-mastoid foramen (extracranial). Cortical facial paralysis is seen following injuries, tumors, abscesses, and localized inflammations of the motor centres. Intracerebral cases result from syphilis of the vessels or hemorrhage. The nuclear form, originating in the pons, is found in Duchenne's disease and in some cases of tabes. These three forms usually present normal electrical excitability and the retention of ability to use the occipito-frontalis and orbicularis palpebrarum muscles. On the contrary, when the lesion is within the bone or beyond it, all three divisions of the facial are equally involved. Hence the presence of lagophthalmos is a valuable diagnostic sign. Injury to the facial nerve in the aqueduct of Fallopius is not an uncommon sequel to middle-ear suppuration. The site of the lesion can be determined by the presence or absence of such symptoms as paralysis of the soft palate, diminution or abnormal acuteness of hearing, diminution in the saliva, or alterations in taste. Beyond the stylo-mastoid foramen the nerve can suffer from injuries either accidental or surgical, the growth of tumors or enlarged glands, and certain indefinite causes, attributed to taking cold. It is evident that the prognosis of paralytic lagophthalmos must be much more unfavorable if the lesion be central than if peripheral. Usually the so-called rheumatic facial paralysis lasts only a few weeks. The electrical condition of the affected muscles will furnish valuable data. If there is no change in their reaction to either electric current, the prognosis will be favorable; if the "reaction of degeneration" is found, recovery will be slow; if secondary contractures and twitchings supervene, the recovery will either be very slow or will never occur (Ramsay).

Cases in which lagophthalmos is a symptom of disease of the third nerve are of rare occurrence. Shortening of the eyelids leads to exposure of the globe and, when congenital, is named congenital lagophthalmos. When acquired it may be produced by laceration of tissue, burns or scalds, necrosis of the osseous or gangrene of the soft tissues, etc.

Treatment.—This will vary according to the cause. The conjunctiva should be kept clean by frequent instillations of a solution of boric acid. In exhausting diseases the lids should be kept closed by adhesive plaster or bandages. In severe cases of lagophthalmos, where the cause is irremediable, the operation of uniting the lids (tarsorrhaphy) should be performed. This unites the lids, shortens the palpebral fissure, and protects the cornea.

Blepharophimosis, a narrowing of the palpebral openings, is found in congenital and acquired forms. The former is rare; the latter compara-

tively frequent, being caused by trachoma. In unusual cases it is due to loss of tissue and consequent contraction at the outer canthus. It is properly treated by canthotomy.

Blepharospasm, a tonic or clonic spasm of the orbicularis palpebrarum muscle, firmly closing the lids, is a common symptom in conjunctival and corneal injuries and diseases (symptomatic blepharospasm). It not infrequently occurs without known pathologic condition (essential blepharospasm). In the tonic form the lids close spasmodically and remain closed for a time; in the clonic form the spasm is of short duration, is immediately followed by relaxation, and this in turn is followed by another spasm. A common form of blepharospasm is the fibrillary contractions, which are often alarming to the patient. The twitching of the fibres can be seen near the lid-margin. It indicates a local irritation, such as often is found in mild conjunctivitis or in errors of refraction, but often is without significance. It is frequent in habit chorea and hysteria. These clonic conditions are of momentary duration. In some cases clonic blepharospasm is a distressing affection, particularly in aged subjects. The contractions are frequent and violent, involving not only the orbicularis, but also the adjacent facial and temporal muscles. This form is often rebellious to treatment. Hysteric blepharospasm is often seen in neurotic females up to the middle period of life. Von Graefe described cases of spasm of the orbicularis occurring in persons who had been subject to trigeminal neuralgia, the supra-orbital and supramaxillary branches being often involved. The tonic contraction in these cases can be controlled temporarily by pressure over the nerve-branches or permanently by subcutaneous section of the trunk. Similar cases and results are seen in persons who have never been subject to neuralgia. By far the most frequent class of cases is that in which the spasmodic action follows irritation or inflammation of the conjunctiva, lid-margin, or cornea (reflex blepharospasm). It is present in practically all cases of phlyctenular conjunctivitis and keratitis and often persists long after the subsidence of the acute conjunctival or corneal symptoms. In such cases careful examination will show the presence of a minute fissure located more often at the outer than at the inner canthus. The patient with reflex blepharospasm shuts the eyelids tightly, strenuously resists efforts to open them, hides the head in a handkerchief or pillow, and dreads exposure to light.

A rare and obscure form of disease is that in which persistent tonic blepharospasm occurs. When finally the eyes are opened, there may be temporary loss of vision without fundus changes, or great reduction in visual acuity with marked retinal, chorioidal, or optic-nerve lesions.

TREATMENT.—In all cases of acute blepharospasm search is to be made for the cause, which, if possible, is to be removed. Blepharitis marginalis, follicular conjunctivitis, phlyctenular keratitis, fissure of the canthus, ametropia, insufficiency of the external ocular muscles, carious teeth, nasal and pharyngeal lesions, anemia, and chlorosis are among the conditions

frequently found. Appropriate local treatment, together with the internal use of arsenical and ferruginous preparations, will generally bring about a cure. The ancient and barbarous method of seizing the child by the heels and immersing the head in cold water, in order to overcome blepharospasm, is no longer practiced. In rebellious cases of fissure the lids are to be stretched, the fissure cauterized with solid stick of nitrate of silver, and the ointment of yellow oxid or ammoniated mercury applied. This severe treatment should be followed by cure, provided the conjunctival and corneal affections are properly treated. In all cases of spasm search is to be made for foreign bodies in the fornices and cornea. Hysteric blepharospasm is best treated by suggestion. In senile blepharospasm treatment will likely be beneficial if pressure-points can be found. Hypodermic injections of morphin or cocain, the use of the galvanic current, or the application of veratrin ointment are suitable remedies. Large doses of the fluid extracts of conium and gelsemium may give relief. Stretching of the trunk of the facial nerve, while sometimes successful, is generally without permanent value.

Trichiasis and Distichiasis.—In these conditions (Fig. 1, Plate VII) the cilia are turned inward and rub against the globe. In trichiasis the lashes turn inward without inversion (entropion) of the lid, the condition being the result of long-continued blepharitis or trachoma. In entropion the lid-margin is inverted, the tarsal plate being deformed. The term distichiasis is applied to a condition in which supernumerary cilia are present. Commonly the cilia are present in two rows, one of which is directed normally, the other being turned backward and rubbing the globe. Often small white hairs, best discovered by the aid of oblique illumination or by the use of the binocular magnifier, are found along the lid-margin or in the inner canthus. When directed toward the globe or toward the caruncle they cause a persistent conjunctivitis.

The rubbing of the cilia against the eyeball causes a constant sensation of sand or foreign body, and leads to redness, lacrimation, conjunctival discharge, and vascular keratitis. Under the circumstances, it is useless to expect benefit from remedies applied to the conjunctiva or cornea until after the source of irritation has been removed.

TREATMENT.—If the misplaced cilia are few in number, they should be pulled from time to time until the patient is willing to submit to operative intervention. The conditions mentioned above can be relieved by electrolysis: a sure, but painful and tedious, method. When numerous cilia are misdirected, a suitable operation should be performed.

Symblepharon is described in the chapter on diseases of the conjunctiva.

Ankyloblepharon, adhesion of the eyelids along the palpebral margin, may be partial or total. It results from the growing together of two raw surfaces. It is rarely congenital. As an acquired affection it follows ulcerations, burns, and other injuries. It is sometimes observed after

croupous conjunctivitis. Ankyloblepharon may exist alone, but more frequently symblepharon also is present. The prognosis is favorable in uncomplicated ankyloblepharon, an operation (cutting the adhesions) serving to restore the palpebral opening. When associated with symblepharon the case will require also one of the symblepharon operations.

Ptosis (Blepharoptosis), a drooping of the upper eyelid, may be partial or complete, unilateral or bilateral, congenital, hereditary, or acquired. In the higher degrees it interferes with vision, the lid covering the cornea. In bilateral cases the patient will throw the head far back to gain better vision. At the same time the occipito-frontalis muscle is brought into play. Thus a picture is presented which is characteristic. In true ptosis the levator of the upper lid is either congenitally absent, injured, or its nerve-supply—a branch of the third nerve—is interfered with. Spurious ptosis is seen in patients with tumors and inflammations of the upper lid. Unilateral ptosis, dating from birth, may be due to injuries during instrumental delivery. There are interesting cases of unilateral ptosis in which, while the eye cannot be opened by the strongest effort of the will, it will immediately open with the opening of the mouth. Thus there is associated action of the levator and pterygoid muscles. In other cases the upper lid can be raised only when the eye is adducted or abducted. Paralytic ptosis may exist alone or in association with paralysis of the other ocular nerves, notably the fourth and sixth nerves. It is a part of the symptom-complex of the rare disease known as recurrent oculomotor paralysis (ophthalmic migraine of Charcot). It is a striking symptom in ophthalmoplegia externa or chronic nuclear palsy. It may be a symptom of disease in the cerebral hemisphere. According to Swanzy, it is often the result of a cortical lesion. Steffen saw a case of double ptosis due to tubercular degeneration of the corpora quadrigemina. It is found sometimes in crossed hemiplegia, the lesion being intrapeduncular. Ptosis is common in hysteria and congenital ataxia.

Anemic women, at or about the menopause, are subject to a form of the disease, which, from its generally appearing upon awakening from a long sleep, is known as matinal ptosis. Slight degrees of ptosis are found in paralysis of Müller's muscle from disease or injury of the sympathetic nerve or from the intentional removal of the superior cervical sympathetic ganglion. In these cases miosis, unilateral sweating, and diminished intraocular tension are prominent symptoms. Ramsay has recorded a case of reflex ptosis in which a cure speedily followed the extraction of a decayed molar tooth. In elderly persons slight ptosis is sometimes associated with uncorrected presbyopia.

TREATMENT.—Every case of ptosis should be studied carefully to determine its cause. Antisyphilitic or antirheumatic remedies will cure many cases. Electrical treatment is of some value. After these measures have failed and the condition is of long standing, one of the operations described elsewhere in this chapter should be performed.

Ectropion is an eversion of the eyelid. This, of course, exposes the conjunctiva. The condition (Fig. 5, Plate VII), which may be partial or complete, has been classified by Czermak as follows:—

1. Ectropion from traction on the anterior part of the lid: *(a)* cicatricial ectropion, and *(b)* ectropion from division of the lid by a wound vertical or oblique to the musculature (the so-called wound-coloboma).

2. Ectropion from relaxation of the lid-margin: *(a)* in paralysis of the orbicularis muscle (paralytic ectropion), and *(b)* in relaxation of the tissues and loss of the muscle-tone of the palpebral portion of the orbicularis (the ectropion of chronic conjunctivitis, chronic inflammation of the lid-margins, and of senility).

Fig. 153.—Cicatricial ectropion. (Author.)

3. Ectropion from malposition of the tissues in consequence of pressure on the lid-margin from behind, or from backward traction on the convex tarsal surface: *(a)* spastic ectropion, *(b)* in ectasiæ or tumors of the anterior part of the globe with exophthalmos, *(c)* from pressure on the lower lid by conjunctival and tarsal tumors, *(d)* from traction of tumors on the peripheral part of the tarsus or on the conjunctiva. These are cases of mechanical ectropion.

The symptoms of ectropion are the turning out of the lid, epiphora, and thickening of the conjunctiva. The skin of the cheek is eczematous. If the ectropion is slight, it causes so little discomfort that many persons do not seek relief. If the eversion is great, corneal complications are likely.

Treatment.—The treatment of ectropion is operative, and will be considered in the latter part of this chapter.

Entropion, a turning in of the lid, is either muscular or organic. The former is seen as a result of bandaging in elderly persons whose eyes lie deeply in the orbits. It occurs also in infants at birth, from excessive development of the orbicularis muscle; and is found at different ages in spasmodic form from irritation accompanying conjunctivitis, keratitis, and the lodgment of foreign bodies. Organic entropion is commonly caused by trachoma. It also follows diphtheritic conjunctivitis and essential shrinking of the conjunctiva (Fig. 6, Plate VII).

The effect of entropion is to place the skin of the lid in contact with the globe of the eye. The lashes constantly rub against the cornea and produce characteristic changes in that tissue. The epithelium becomes abraded and the deeper layers of the cornea become necrotic. Pannus develops and vision is much reduced.

TREATMENT.—The muscular or spastic form of entropion generally improves with the removal of the cause. In infants the application of collodion to the outer surface of the lid will be beneficial, or the inversion may be overcome by adhesive plaster. Organic entropion, so often associated with trichiasis and blepharophimosis, requires operative treatment.

INJURIES OF THE EYELIDS.

Wounds of the Lids may be punctured, incised, lacerated, or contused. Punctured wounds are of little importance provided other ocular structures are not injured. They generally heal without scars. It must be remembered that numerous cases are recorded in which foreign bodies, after traversing the lids, have lodged and remained in the orbit without producing acute symptoms. Incised and lacerated wounds call for careful attention. They should be cleansed and accurately approximated with catgut sutures. It is especially important to note whether the canaliculus has been cut or the globe injured. Horizontal cuts do little harm except the suspensory ligament of the upper lid is severed. Vertical and oblique incised wounds, unless seen early and properly sutured, will lead to coloboma, ectropion, entropion, or trichiasis. When the internal palpebral ligament and canaliculi are cut, the function of the lacrimal apparatus will be interfered with. Lacerated wounds, often produced by bursting bottles, meat-hooks, blows, or thrusts with pieces of wood or a cow's horn, button-hooks, etc., if treated early will generally give good results. If the canaliculus is torn, the remaining portion should be sought and opened into the sac. It may be possible to unite the two portions by passing a short probe, suturing the lid upon it, and leaving the instrument in place for a few days. Each case of laceration must be judged by itself, and often the ingenuity of the surgeon will be taxed.

Contused wounds of the lids, frequent in persons pugilistically inclined, are followed almost immediately by extravasation of blood into the cellular tissue, producing a condition commonly called "black eye." The blood may be in the form of a diffused ecchymosis or as a hematoma. For prognostic

reasons, it is important to distinguish between such an immediate ecchymosis and that which, occurring in fractures of the base of the skull or rupture of orbital vessels, appears later. In such serious injuries, the blood not infrequently is forced forward into the eyelids. The lower part of the ocular conjunctiva and the lower eyelid (rarely the upper lid also) show hemorrhages. The ordinary "black eye" disappears in two or three weeks. If a fracture has involved the frontal or ethmoidal sinus, emphysema, occurring early, may be associated with a tardy ecchymosis. Edema of the lids is a common result of a blow.

TREATMENT.—A "black eye" should be bathed with cold water and treated with frequent applications of arnica, lead-water, laudanum, or hamamelis. If the blood is present as a hematoma, it will be best to incise the lid and evacuate the clots under aseptic precautions. Abscess of the

Fig. 154.—Burn of face and eyelids. (VAIL.)

lid should be treated by incision and the frequent use of a bichlorid solution. Leeches are of no particular value in the treatment of black eye. In sensitive persons the surgeon may conceal the injury by painting the eyelids.

Burns and Scalds of the Eyelids, if of the first or second degrees, generally heal without deformity. Deeper lesions are frequently followed by cicatricial contraction, displacement of the lid-borders (ectropion), ankyloblepharon, or symblepharon. These conditions will require appropriate surgical treatment. When called to a case of burn or scald, involving the lids, the surgeon should note carefully the condition of the conjunctiva and cornea. The injured area should be treated with gauze soaked in carron-oil, or with lint soaked in a solution of borax or sodium bicarbonate, or painted daily with white lead. Iodoform may be dusted on the surface daily. Large granulating surfaces should be covered with Thiersch's skin-grafts. The principles which guide the surgeon in the treatment of burns

and scalds elsewhere in the body will apply to lid injuries. Pain may be so severe as to call for the use of morphin.

Foreign Bodies in the Eyelids. — With the exception of grains of powder, the retention of foreign bodies within the eyelids is of comparatively rare occurrence. Pieces of iron, steel, gun-caps, coal, pencils, splinters of wood, birdshot, dirt, sand, and pebbles are among the substances occasionally found in the lids. Most foreign bodies carry infection with them and produce localized abscesses. In the course of the inflammation the foreign body often is extruded. Metallic bodies, which have been propelled by an explosion, are often sterile and remain imbedded in the lids without causing reaction. Large foreign bodies can be removed through suitable incisions. Small ones can be picked out with a cataract-needle. If the patient is seen shortly after the accident, grains of powder can be removed by scrubbing with a nail-brush. The continued use of gauze soaked in hydrogen peroxid, applied soon after the accident, is said to be of value. If these measures fail, the surgeon should wait until the acute stage has passed, when the

Fig. 155.—Result of burn of face and eyelids. (Vail.)

individual grains may be picked out through small incisions or destroyed with the fine point of an electric cautery (Jackson).

OPERATIONS ON THE EYELIDS.

The eyelid and conjunctiva and adjacent parts (skin of the cheek, forehead, etc.) should be made surgically clean, as is described in the chapter on preparation for ophthalmic operations. All instruments and dressings should be sterile. The surgeon and his assistants should thoroughly clean their hands. Lid operations can be performed, as a rule, under local anesthesia. A solution of cocain (2- to 4-per-cent. strength) is to be dropped into the conjunctival sac as well as injected beneath the skin of the eyelid. General anesthesia will be required for children, and for some of the extensive plastic operations on adults.

Chalazion Operations.—In operating on chalazion, a lid clamp should be used to control hemorrhage and give a solid substance on which to cut. The author has modified Snellen's clamp (Fig. 156), and finds the new modification an admirable instrument. The incision can be made from the skin or conjunctival surface. If the surgeon is simply to incise and curette

the cyst, the incision should be conjunctival; if he desires to excise the tumor, it will be best to make the cut in the skin parallel with the lid-margin. After excising the chalazion the wound is closed with two or three fine sutures. If the conjunctival incision is employed sutures are not necessary. Some surgeons incise chalazia and remove the contents by curettement. If the chalazion is situated near the lid-margin a neat operation is this: The surgeon grasps the lid with the thumb and index finger of the left hand, making traction in the direction of the lid-margin. Then the margin is incised with a von Graefe knife. Through this opening a curette is introduced and the tumor is removed by its use.

Operations for Ptosis.—These are numerous, and many of them are inefficient. Cases of ptosis are divisible into: 1. Those in which the condition is congenital and the levator is absent or imperfectly developed. 2. Those due to defective development of the nerve-centre of the levator, to which class belong those cases of congenital ptosis that can lift the upper lid only in association with certain movements of the jaw. 3. Those of acquired complete paralytic ptosis. 4. Those in which the upper lid droops

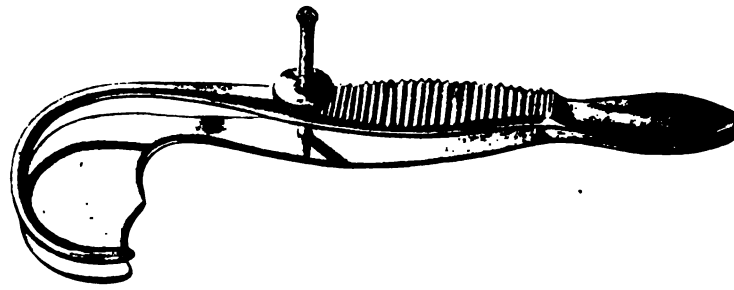


Fig. 156.—Entropion forceps. (AUTHOR.)

after trachoma or the growth of tumors in the lid (hypertrophic ptosis or ptosis adiposa). 5. Those which are due to trauma.

It might seem that shortening of the levator of the upper lid would suffice to correct ptosis, but such procedures have generally given poor results, and are of value only in cases of traumatic separation of the levator from the tarsus. It is only in the cases of slight ptosis that the ingenious procedure of Eversbusch (doubling of the levator muscle over the tarsus) is applicable. *Von Graefe's operation* of excising an elliptical portion of the skin and underlying tissues down to the tarsus is almost valueless, since in most cases either the deformity soon returns or the cornea is insufficiently covered. Operations which aim to unite the occipito-frontalis muscle and the lid are ingenious, and some of them are of value. The procedure which the author believes to be the most efficient of all is Wilder's operation, which aims to lift up the lid so that the eye can be used and at the same time permits the lids to close at will. It consists in folding the tarso-orbital fascia, as well as the aponeurosis of the levator muscle, upon itself. In performing this operation an incision two inches long is made in and

parallel with the eyebrow. "A retractor being used to draw down the lower lip of the wound, the skin and muscle are separated from the fascia by careful dissection until the tarsus is brought into view. This is more easily accomplished if an assistant puts the lid on the stretch. Sutures of fine sterilized catgut or silk, armed at each end with a curved needle, are passed into the tarsus to secure a firm hold at a point about at the junction of the outer and middle third and a little distance from its convex edge (*o* in Fig. 157). It is then drawn through and with it several gathering stitches *(u)* are taken in the tarso-orbital fascia, after which the needle is made to pass through the muscle and connective tissue of the upper lip of the wound. Another needle, on the same suture, follows a parallel course in the same manner, entering the tarsus about three millimetres from the point of entrance of the first, then gathering the fascia into small folds and emerging in the tissue above, thus making a loop by which the lid may be drawn up. A second suture is passed in the same way, making a

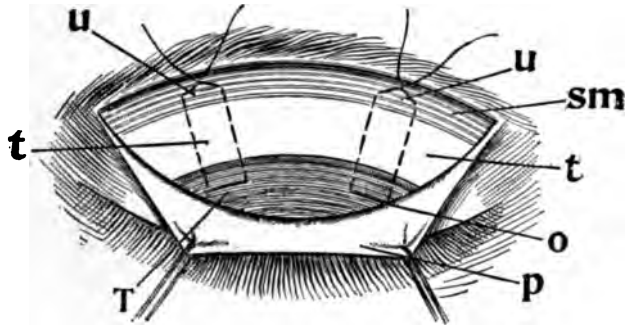


Fig. 157.—Wilder's operation for ptosis.

T, Tarsus. *t*, *t*, Tarso-orbital fascia. *sm*, Supra-orbital margin. *p*, Lower lip of wound drawn down. *u*, *u*, Sutures.

loop at the junction of the middle and inner third of the tarsus. The requisite elevation of the lid may now be secured by drawing on the loops and tying the sutures which are to be buried in the wound." The author can confirm Wilder's statement that "complete ptosis may be relieved by shortening the suspensory ligament of the lid with buried sutures."

PANAS'S OPERATION, which can be understood by examination of the accompanying illustrations, has been extensively practiced with fair results. The objections to it are that, in placing the rectangular flap under the bridge, a skin surface is opposed to raw tissue. In healing a pouch is left in which dirt accumulates, and in some cases the growth of hair from the buried flap causes annoyance. This operation raises the tarsus and permits the occipito-frontalis muscle to discharge in large part the functions of the absent or paralyzed levator.

Van Fleet and Allport have modified Panas's operation.

The principles of the von Graefe and Panas operations have been combined in *Tansley's procedure,* which is performed in this manner: The

surgeon makes two perpendicular cuts (Fig. 160, *A, B, C, D*) one-fourth of an inch apart, and extending from the upper orbital margin to within two lines of the edge of the upper lid. These are united at the upper extremity by a horizontal incision, *A-C,* and then the ribbon of tissue is dissected and permitted to drop down upon a wad of cotton lying upon the cheek, which is kept moistened with warm Panas's solution.

Then a curved cut is made from *H* to *G* and *E* to *F,* following the crease which shows the upper limit of the tarsal cartilage, and a straight cut is made from *H* to *B* and from *D* to *F,* parallel to and about four millimetres distant from the lower border of the upper lid. The derma and the orbicularis muscle, which are embraced within these cuts, are then carefully dissected off, leaving the whole tarsal plate denuded of tissue. This denuded surface is carried a trifle beyond both the internal and external canthi. The cut edges *H-G* and *E-F* are united to the cut edges *H-B* and *D-F,* respectively, by interrupted sutures. Then a von Graefe knife is

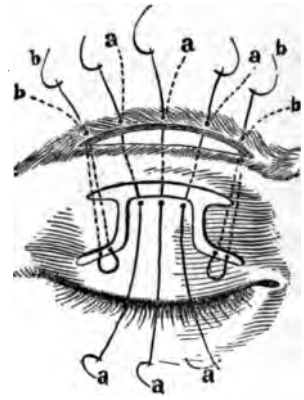


Fig. 158.—Incisions and sutures in Panas's operation.

entered at *A-C* and passed beneath and brought out upon the forehead just above the eyebrow, and slight lateral cuttings are made so as to give room for the passage of the ribbon of derma which has been dissected up at the first stage of the operation. Then passing a strong suture into the upper edge of this ribbon, it is drawn up into the cut made beneath the eyebrow, and is brought out upon the forehead. When drawn up sufficiently tight, so as to leave no folds of tissue or puckerings, it is cut off smooth with the forehead and fastened there by two small sutures. Then several sutures are placed from *A* to *G* and *C* to *E,* uniting the edges of the ribbon to the bordering derma.

The relief of ptosis by a permanent wire suture is the feature of *Mules's operation.* Its author described the procedure as follows: "Two needles with eyes near their points were passed deeply through the frontalis tendon over the eyebrow, and their points brought out at the margin of the lid behind the lashes, taking up a substantial part of the tarsal cartilage

on their way. A piece of silver wire was threaded through each needle, which was then withdrawn, leaving the loop of wire passing from the brow to the edge of the lid and back to the brow again. This was then tightened until the lid was sufficiently raised, the edge of the lid being grooved by an incision to allow the wire to sink into the substance of the lid. One end of the wire was then passed under the skin and made to emerge by the side of the other end of the wire. The two ends of the wire were then

Fig. 159.—Appearance of the eye after Panas's operation.

twisted on each other until the lid was raised permanently, the ends were cut off, and the wire allowed to sink below the level of the skin. The skin at this point and the lid-margin healed over the wire, which remained permanently fixed in the substance of the lid. From further experience it was found that the wire remained in position without causing irritation; the lids could be closed, and remain closed during sleep. All kinds of wire had been tried, but it had been found that silver wire was the most satisfactory.

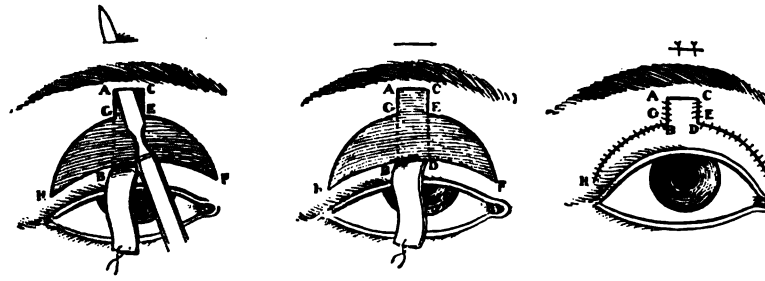


Fig. 160.—Operation for ptosis. (TANSLEY.)

It was necessary to note at the time of the operation the situation of the twisted end of the wire in case it became necessary to remove the suture afterward."

The ptosis remaining after trachoma can best be corrected by sutures introduced in the manner similar to Hotz's operation for entropion. Other ptosis operations, which can be mentioned only by name, have been devised by Snellen, Eversbusch, Wolff, Hess, Pagenstecher, Birnbacher, Dransart, Kunn, Motais, Parinaud, and Gillet de Grandmont.

Tarsorrhaphy.—This operation is performed for the purpose of narrowing the palpebral fissure, and is done for the following conditions:—

1. In lagophthalmos, when the cornea is partly uncovered during sleep and consequently is liable to necrosis. The condition may be due to congenital malformations of the lid (ablepharia), paralysis of the orbicularis muscle, or to traumatic lesions of the lids.

2. In exophthalmos, particularly that of exophthalmic goitre.

3. In ectropion of any form, and particularly in that of the paralytic variety.

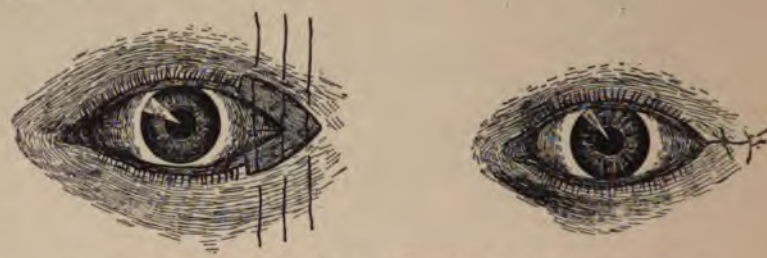


Fig. 161.—Tarsorrhaphy by von Graefe's method. (CZERMAK.)

4. In plastic operations.

5. In excision of the Gasserian ganglion tarsorrhaphy is done beforehand to prevent corneal ulceration.

Tarsorrhaphy may be external, internal, median, or total. External tarsorrhaphy can be done by either the method of *von Graefe* or that of *Fuchs*. In the former a sufficient amount of the outer end of each lid-margin is denuded and sutures are passed as shown in Fig. 161. In Fuchs's

Fig. 162.—Tarsorrhaphy by Fuchs's method. (CZERMAK.)

operation (Fig. 162) an incision, made in the intermarginal line, separates the cutaneous part of the lower lid from the tarsus. On the upper lid an area of corresponding extent is denuded; then by means of a single suture the parts are brought together. In median tarsorrhaphy the denuded surfaces are situated on the central part of the lids.

Canthoplasty (Blepharotomy).—The operation of canthoplasty, tarsodialysis, or blepharotomy, is made for the purpose of enlarging a palpebral opening which is abnormally small. The most common cause of this condition is trachoma. Congenital smallness of the palpebral opening may

be an hereditary characteristic. In almost all operations for entropion a canthoplasty, or at least a canthotomy, must be made before the lid-clamp can be applied and the entropion properly treated. In this case the canthotomy is simply a part of the operative procedure for entropion. In making a canthoplasty a local anesthetic should be dropped on to the

Fig. 163.—Von Ammon's canthoplasty. (After CZERMAK.)

conjunctiva as well as injected beneath the skin at the outer canthus. The instruments needed are a pair of straight scissors, a needle, a needle-holder, and forceps. The surgeon introduces one blade of the scissors beneath the outer canthus, and cuts outward to the malar bone or to a

Fig. 164.—The sutures in place in canthoplasty.
(After CZERMAK.)

less extent. The conjunctiva and skin are then to be united by sutures and an appropriate dressing is applied.

Trichiasis Operations.—When only a few cilia are at fault, they may be removed from time to time with cilium forceps, or the follicles can be destroyed by electrolysis according to the method of Michel, of St. Louis. The latter is a slow and painful procedure. In these cases the affected part of the lid should be transplanted. A straight keratome is introduced at

the intermarginal line, and is made to split the lid into two parts, the hair-follicles being in the flap in front of the tarsus. The operation can then be finished in one of two ways: either the part bearing the faulty cilia can be excised by removing a triangular segment of skin, as is done by Stephenson, or, as the author prefers, a button of skin can be removed, the lid-border and cilia being transplanted. The latter procedure is to be preferred. The wound is to be closed with catgut sutures. Desmarres, Sr., treated these cases by the excision of a small oval piece of skin and trusted to cicatrization to draw the cilia away from the globe.

In case the greater part of the eyelid is involved in trichiasis, a general transplantation of cilia must be done. For the relief of this condition no procedure is so valuable as a properly-made *Jaesche-Arlt operation.* In patients with trichiasis it is always wise to permit the cilia to be undisturbed for at least a week before operation, in order that the surgeon shall locate them without difficulty. The instruments needed are a lid-clamp, a

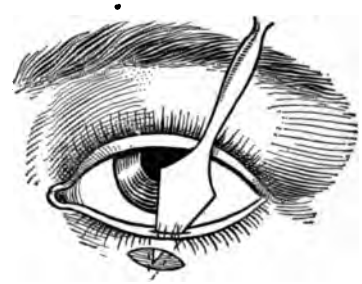


Fig. 165.—Excision operation for trichiasis. (After STEPHENSON.)
The dotted lines show the parts removed in the excision operation; the oval marks the tissues removed in the author's method.

scalpel, fixation-forceps, needles, needle-holder, scissors, and a sharp and strong von Graefe or Beer knife for splitting the lid. The first step is the separation of the lid into two parts: an anterior, containing the skin, muscular fibres, and cilia; and a posterior, composed of tarsus and conjunctiva. To do this the surgeon grasps the everted lid between the left thumb and index finger, and passes the knife to the depth of 3 or 4 millimetres along the intermarginal line from near the punctum to the outer canthus. To make this incision properly is sometimes difficult, yet its correct performance has much to do with the success of the operation. The lid-clamp then being applied, an elliptical piece of skin and other tissues is removed down to the tarsus. The next step is to connect the intermarginal incision with the wound on the outer surface of the lid, thus making a bridge which consists of skin and orbicularis muscle, free above and below, but attached at either end. This bridge is then to be sutured to the upper edge of the lid-wound. The result is that the bridge bearing the cilia is moved away from the lid-margin and the hairs are given a normal direction. To prevent cicatricial contraction some surgeons use

the excised piece of skin to cover the raw surface of the tarsus. Others transplant a piece of the mucous membrane of the mouth into the wound. These devices are not necessary. The sutures are to be removed in four or five days. The lid will remain thickened for several weeks. Sloughing of the bridge has never occurred in the author's practice.

Some surgeons make the intermarginal incision in this way: Jaeger's lid-plate is placed under the lid and a keratome is used to make the incision, the lid being supported by an assistant.

Entropion Operations.—Of the many procedures for the relief of entropion, *Snellen's operation* has probably given the most satisfactory results. It is applicable to cases of organic entropion: *i.e.*, to those in which the tarsus is incurved. The instruments needed are the lid-clamp, scalpel, tissue-forceps, needles, needle-holder, suture material, and a half-dozen beads. The clamp having been applied, an incision 4 millimetres from the lid-margin is to be made along the length of the upper lid. A

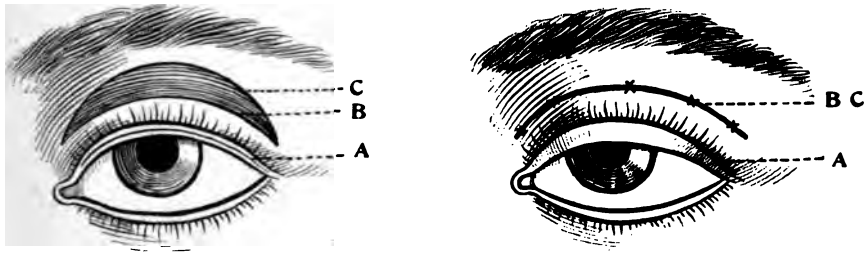


Fig. 166.—Jaesche-Arlt operation for trichiasis.
The figure at the left shows the incisions, *A* being the cut in the intermarginal line, and *B*, *C*, the skin removed.

second incision, 2 or 3 millimetres higher, connects with the first cut. The skin and orbicularis muscle included between these incisions are to be removed, thus exposing the tarsus. The surgeon should pass his finger along the tarsal plate and notice the place of greatest convexity, from which a wedge-shaped segment is to be excised. To do this properly requires a very sharp scalpel or a Beer cataract-knife. The long diameter of the excised wedge is to be parallel with the lid-margin. The next step, the insertion of the sutures, is of importance, and is often improperly described. The sutures are usually three in number, each one being armed with a needle at each end. One needle is to be passed into the upper segment of the tarsal tissue parallel with the wound, so as to include about 2 or 3 millimetres of tissue. The two needles of this suture are now to be passed through the lower segment of the lid, 5 millimetres apart, in this manner: Grasping the lid-margin with tissue-forceps, the surgeon passes each needle from the point *G* (Fig. 167) to *G*. This procedure is to be repeated with the two other sutures. Then the lid-clamp is to be removed and a bead is to be strung on each end of each suture, the sutures being tied and the ends

left long. The ends are then drawn taut against the forehead, thus everting the lid. They are to be fastened in this position by strong adhesive plaster strips. There will be no need for suturing the skin in this operation, since the eversion which follows the fastening of the sutures to the forehead causes the lips of the skin-wound to approximate. A dry gauze dressing is to be applied. The sutures are to be left attached to the forehead for two days,

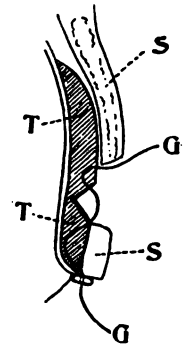


Fig. 167.—Section of the upper eyelid in Snellen's operation for entropion.
S, S, Skin. *T, T*, Tarsus from which a wedge has been cut. *G, G*, Suture.

after which the ends are to be cut off and the lid is permitted to assume its proper position. On the fourth day the beads and deeper part of the sutures are to be removed.

For obvious reasons Snellen's operation is applicable only to the upper lid. In case the lower lid is incurved, *Hotz's operation* should be performed. A spatula having been placed under the lid, an incision 4 milli-

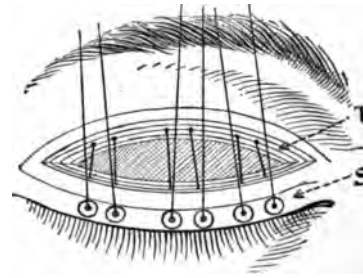


Fig. 168.—The method of placing the sutures in Snellen's operation for entropion.
T, Tarsus. *S*, Skin.

metres removed from, and parallel with the lid-margin is to be made, and narrow pieces of skin and orbicularis muscle are to be removed. This exposes the lower margin of the tarsus. If it does not come promptly into view, the edges of the wound are to be held apart and the soft tissues are to be dissected down to the tarsus. All muscular fibres adhering to the lower third of the tarsus must be removed. A needle is to be passed through the palpebral skin; then it is carried through the bared edge of the tarsus

into the tarso-orbital fascia for a short distance, and is then passed through the lower edge of the incision without including any muscular fibres. Two other sutures are to be used in a similar manner. When tied, the sutures bring the skin into contact with the tarsus, to which it adheres. Thus

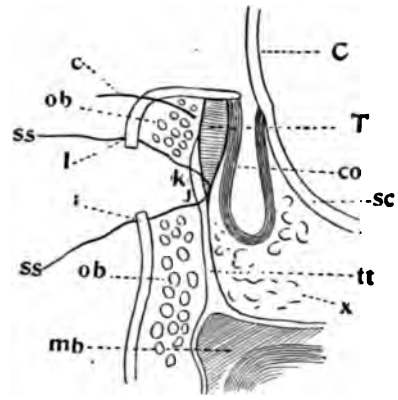


Fig. 169.—Hotz's operation for entropion.

C, Cornea. *T*, Tarsus. *co*, Conjunctiva. *sc*, Sclera. *tt*, Tarso-orbital fascia. *x*, Orbital fat. *mb*, Malar bone. *ob*, Orbicularis muscle. *ss*, Suture. *c*, Cilium. *l*, *i*, Lips of wound.

the tarsus is made the fulcrum: a point which Hotz emphasizes. This operation is applicable to either lid. It is proper to state that an operation founded on the same principle as that of the Hotz procedure was described by Anagnostakis in 1857.

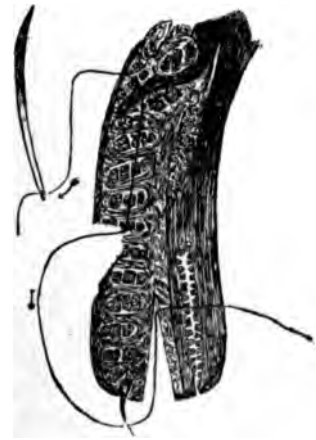


Fig. 170.—Harlan's operation for entropion.

HARLAN'S OPERATION.—The lid is to be split in the ordinary manner and a horizontal strip of skin and muscle is removed. The anterior segment of the lower half of the lid is not converted into a bridge. The sutures are passed through the posterior segment of the lid, then looped over the

anterior segment, and the needle is passed deep into the orbicularis muscle, emerging 5 millimetres above the wound. When tied, the sutures draw the tarsus and conjunctiva downward and the cilia-bearing segment forward.

In cases of spastic entropion the *thread operation of Snellen* is useful. The object is to draw the margin of the lid away from the globe and to form in the lid cicatricial bands which shall continue to maintain it in proper position after the sutures have been withdrawn. A thread, armed with two needles, is to be passed through the deepest part of the lower fornix and is brought out through the skin; then each needle is reintroduced and is made to pierce the full extent of the lid, being brought out at the outer lip of the lid-margin. The threads are then tied tightly over a small roll of gauze. Two or three such threads are used. The points of exit on the lid-margin are to be the same distance apart as the space separating the threads in the fornix. This operation is chiefly of use in entropion of the

Fig. 171.—Von Ammon's operation for epicanthus.

lower lid. Other suture operations for spastic entropion have been devised by Gaillard and Arlt.

Ewing's Operation is applicable to atrophic entropion of the lower eyelid. A longitudinal incision is made "through the tarsal conjunctiva and the tarsus, parallel to and from two to three millimetres distant from the line of the opening of the Meibomian glands. This incision, extending through the entire thickness of the tarsal tissue from its nasal to its temporal end, permits the whole marginal strip to be turned forward, to form a new lid-margin of normal width and appearance." The strip is held in place by sutures. The cut on the conjunctival side fills with new tissue, which increases the height of the tarsus two or three millimetres.

Epicanthus Operations.—For the relief of epicanthus the excision of a vertical fold of the redundant tissue from the root of the nose, as practiced by von Ammon early in the last century, has stood the test of time. The tissues are to be lifted up to an extent sufficient to remove the deformity, and on each side a mark is to be made to indicate the location of the incisions. The elliptical area thus outlined is to be excised. The

lips of the wound are to be united with fine sutures (Fig. 171). Every precaution should be taken to secure primary union and avoid an unsightly scar. Wicherkiewicz has recently described an operation which can be readily understood by an examination of Fig. 172.

Recently Broekaert has successfully used paraffin injections beneath the skin over the root of the nose in cases of epicanthus. This treatment is not without danger. Leiser has reported a case in which it caused vomiting, total blindness of the left eye, edema of the lids, hemorrhagic infiltration

Fig. 172.—Wicherkiewicz's operation for epicanthus.
A, After excision. *B*, After the sutures have been tied.

of the iris, and hypotony. These symptoms were attributed to thrombosis of the ophthalmic vein. Hurd and Holden observed embolism of the central retinal artery immediately after a paraffin injection into the nose.

Ectropion Operations. — Acute ectropion results from inflammatory swelling and requires treatment of the accompanying conjunctivitis. In severe cases it may be necessary to scarify the chemotic membrane. In ectropion due to spasm of the orbicularis muscle a canthotomy can be done with benefit. In senile ectropion with simply a falling away from the globe

Fig. 173.—Snellen's thread operation for ectropion.
The figure at the left shows the thread in position to be tied; the figure at the right shows the lid after tying.

of the inner part of the lower lid, slitting of the canaliculus without other treatment will be in order. In cases of senile ectropion of the lower lid, and in the ectropion of children from swelling of the conjunctiva, and with only slight elongation, *Snellen's thread operation* (Fig. 173) is valuable. A stout silk thread, armed with a curved needle at each end, is to be passed into the tissues at the highest point of the everted conjunctiva and brought out upon the cheek. The ends are tied over a roll of gauze and the lid is thus held against the globe. The cicatricial bands which form in the track

of the suture are expected to maintain the lid in position. The suture is to be removed at the end of four or five days.

Where the lid is much elongated and sags to a considerable degree, particularly in cicatricial ectropion, the surgeon has the choice of three procedures: (1) the lid can be supported by the transference of a skin-flap from the malar region, as in von Langenbeck's operation, (2) one of the numerous excision operations can be done, or (3) the Hotz operation can be performed.

Von Langenbeck's Operation, which is highly commended by Bernays, is to be done by making an incision in the skin from one canthus to the other, 2 or 3 millimetres from the lid-margin. The deeper tissues are then to be divided and an assistant pulls the lid into place. The surgeon then makes a skin-flap, of the shape shown in Fig. 174, and sews it into the wound. The skin-flap must be much larger than the space

Fig. 174.—Von Langenbeck's operation for ectropion.
A, Incision. *B*, After suturing.

to be covered, to allow for shrinkage. The redundancy of tissue in the lower lid will disappear.

The excision operations for ectropion are numerous. *Von Ammon's operation* consists in the removal of a triangular segment from the outer canthus, the base of the triangle being toward the eyeball (Fig. 175). *Dieffenbach's operation* combines a tarsorrhaphy with the excision of a triangle, with the base up (Fig. 176), from the outer canthus. *Sanson's*, often misnamed *Adams's, operation* consists in the excision of a wedge-shaped piece of the whole thickness of the lid (Fig. 177).

Robertson's First Operation (Fig. 178) was a modification of Snellen's procedure. A strong silk suture, armed with two needles, is to be passed through the anterior part of the lower lid *(b, b)* and is to be brought out on the conjunctival surface *(a, a)*. Then it is to be passed from the highest point of the eversion downward to emerge on the cheek *(d, d)*. Before tying the suture a piece of lead *(c)* covered with

India-rubber tubing is placed beneath the loops of the suture. The upper end of the rubber-covered piece is to be curved to suit the curve of the eyeball. The lids are then to be closed and the suture tightened. The result

Fig. 175.—Von Ammon's operation for ectropion.

is that the edge of the lid assumes the normal position. The tubing and suture are to be removed at the end of a week. This operation is applicable to senile ectropion of the lower eyelid.

Fig. 176.—Dieffenbach's operation for ectropion.

Robertson's Latest Operation seems to be applicable to several forms of ectropion of the lower lid. An incision is made through the skin of the outer third of the lower lid and is continued upward 12 or

Fig. 177.—Sanson's operation for ectropion.

15 millimetres. It is then to be carried outward 6 millimetres and then downward about 25 or 30 millimetres. The flap of skin thus outlined is to be dissected. A V-shaped piece is next removed from the entire thickness of the lid near the outer canthus. Traction upward is now to be made

on the skin-flap until the lid is placed in normal position. The excess of the skin-flap is cut off and the parts are united with sutures (Fig. 179).

KUHNT'S OPERATION consists in the excision of a triangular piece of the tarsus and conjunctiva, the base of the triangle being at the lid-margin and the apex at the fornix. The wound is closed by means of sutures placed horizontally on the inner surface of the lid, and one external suture. The steps in the operation are shown in Figs. 180 and 181. This procedure

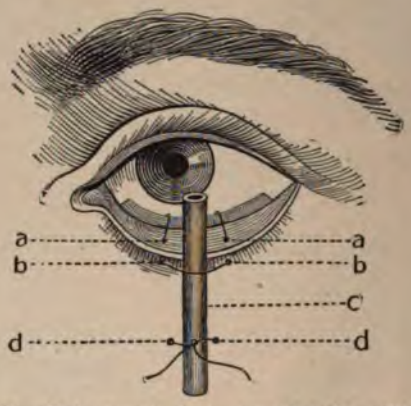


Fig. 178.—Robertson's first operation for ectropion.

is applicable to those cases of ectropion of the lower lid in which the lid is elongated chiefly at the margin. After the sutures are tied, the conjunctiva around the wound is dusted with iodoform, the skin of the lids is anointed with sterile vaselin, and a dressing of sterile gauze is applied. The sutures are removed on the fourth day. Kuhnt's operation is of value in cicatricial ectropion in addition to the required plastic procedure, but, as a rule, it is of greatest value in senile ectropion and in eversion of the lower lid from trachoma.

Fig. 179.—Robertson's latest operation for ectropion.

L. MÜLLER'S OPERATION is a modification of that of Kuhnt. The accompanying illustrations (Fig. 182) make a detailed account of this procedure unnecessary.

HOTZ'S OPERATION FOR CICATRICIAL ECTROPION is to be recommended.

In operating upon the lower eyelid (Fig. 183) the incision, *g-m-b*, is begun 1 centimetre below the inner canthus and is carried obliquely into

the cheek to a point 3 centimetres below the centre of the everted lid-margin. It is then extended upward and outward to the point *b* (Fig. 183), even with and 1 centimetre from the outer canthus. This large flap, *g-m-b*, is dissected from the underlying scar-tissue and all cicatricial bands are cut. The overstretched lid-margin is then reduced to its proper size by

Fig. 180.—Kuhnt's operation for ectropion.

removing a suitable piece (except the conjunctiva) near the outer canthus, cutting from *a* to *c* along the lid-margin and from *c* to *d* through the flap. The edges *c-d* and *a-b* are then united by two silk sutures. The lid is now drawn up as far as possible and is held by two silk sutures, which are passed through the free margin and fastened to the forehead

Fig. 181.—Supporting stitch in Kuhnt's operation.

by adhesive plaster. The edge of the detached lid-flap is then anchored to the tarso-orbital fascia by silk sutures. The open surface is covered with a Thiersch graft whose edges are to lap over the surrounding skin.

In operating on the upper eyelid (Fig. 184) Hotz begins his incision about 5 millimetres above the inner canthus, and continues it obliquely upward into the cicatricial skin and then downward to a point about 5

millimetres above the outer canthus. This incision outlines a large flap, *a-b-c*, which is dissected from the underlying scar-tissue as far as the lid-borders. The lid is then released by dissection from all cicatricial connections until it can be replaced in its normal position. The edge of the lid-flap is then fastened by silk sutures to the upper border of the tarsus. If, on account of the presence of the eyebrows, the lid-flap cannot be taken

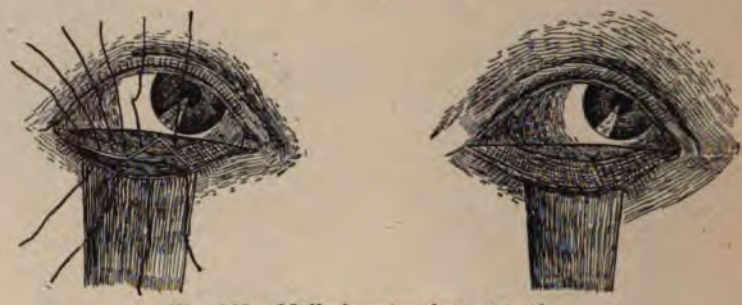


Fig. 182.—Müller's ectropion operation.

from the cicatricial skin, an incision is made along the lid-border. The lid is replaced and a large Thiersch graft is sutured to the upper tarsal border as well as to the wound edge of the free border. In placing the sutures care must be taken that the edges of the flap do not roll in. Strips of gutta-percha are laid over the flap; a gauze compress wet with warm

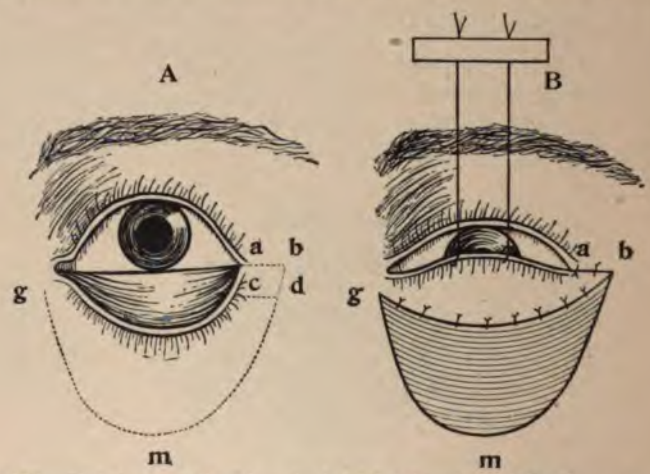


Fig. 183.—Hotz's operation for cicatricial ectropion of the lower eyelid.
A, Lines of incision. *B*, The sutures in place.

boric-acid solution is applied; and a protection layer of cotton and a bandage complete the dressing. The dressing is not disturbed before the third day.

Subcutaneous Incision.—In rare instances cicatricial ectropion can be relieved by the subcutaneous cutting of contracted bands. The operation is done by means of a tenotome.

EXCISION OF THE CICATRIX, without a plastic procedure, is applicable in a few cases where the scar-tissue is of limited extent. In general terms, however, it may be said that cicatricial ectropion demands a blepharoplastic operation.

Blepharoplastic Operations.—These procedures are required in cicatricial ectropion and in cases where destruction of a part or all of the lid occurs. The restoration may be made by grafting or by transplantation

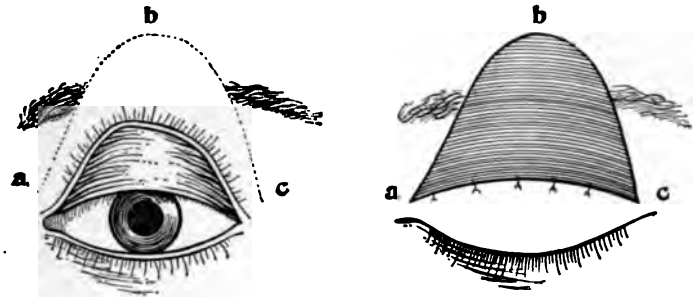


Fig. 184.—Hotz's operation for cicatricial ectropion of the upper eyelid.

of a piece of skin. Such operations are divisible into the following groups:—

1. Operations in which a piece of skin bearing a pedicle is turned into a lid. This may be obtained *(a)* from neighboring tissue or *(b)* from a remote part.

2. Operations in which the transplanted piece of skin is without a pedicle (method of le Fort).

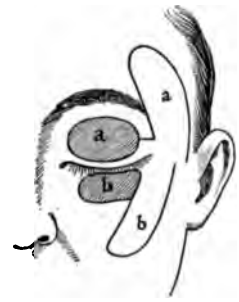


Fig. 185.—Fricke's blepharoplasty.

3. Operations in which small pieces of skin are grafted into a wound as a mosaic (Reverdin's method).

4. Operations in which the grafts of Ollier or of Thiersch are used.

In all blepharoplastic operations the strictest attention to asepsis is necessary. Often repeated operations will be required in order to secure the best result. Tension on the transplanted flap should be avoided, and in cutting the flaps fully 50 per cent. should be allowed for shrinkage. Wherever possible the pedicle should include a good-sized blood-vessel.

Twisting of the pedicle should be avoided. Sutures are used only in limited number; numerous sutures add to the danger of infection. Artificial warmth applied to the flap is unnecessary. A dry dressing should be applied to the parts and the wound should not be touched for two or three days. Syphilitics are not good subjects for plastic operations.

Fig. 186.—Blasius's blepharoplasty.

Blepharoplasty with a Pedicle Derived from Adjacent Tissue.—The methods of blepharoplasty with a pedicle are numerous, and only a few can be mentioned. *Fricke's operation* (1829) consists in filling an oval defect in the lid with a tongue-shaped flap taken from the forehead or cheek (Fig. 185). It is applicable to either lid. Fricke applied this operation to cases of extreme ectropion in which only the skin was destroyed, the conjunctiva and lid-margin being normal.

Fig. 187.—Blasius's blepharoplasty.

Blasius's operation (1842) aims to restore the lower lid by a flap taken from the side of the nose (Fig. 186), not from the glabella and forehead, as has been stated by Meyer and de Wecker. The upper lid, however, can be restored by taking the flap from the root of the nose and the forehead. The same surgeon devised several other ingenious operations, one of which is shown in Fig. 187.

Hasner d'Artha's Operation.—This surgeon devised an ingenious opera-

tion (Fig. 188), by means of sliding flaps with curved margins, for cases in which removal of a large part of both lids becomes necessary.

Dieffenbach's Operation.—In this a triangular defect in the lower lid is covered with a quadrangular flap (Fig. 189). A modification of this operation was made by Szymanowski (Fig. 190).

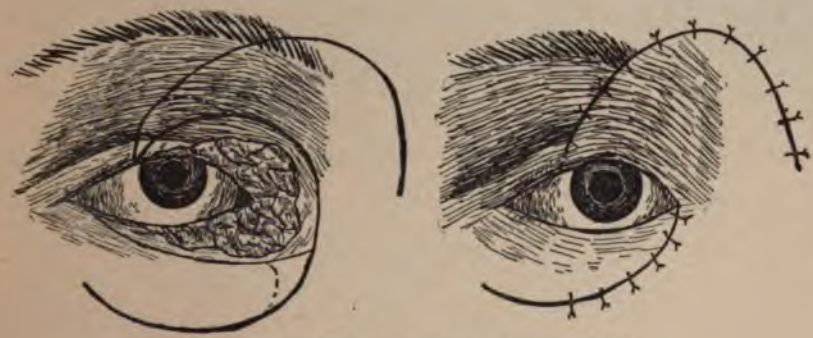


Fig. 188.—D'Artha's blepharoplasty for both lids.

Knapp's Operation (Fig. 191) is valuable where the greater part of the lower lid must be excised for malignant disease. In cases of ulceration where there is involvement of both eyelids, an extensive plastic operation will be required, as in a case reported by Posey and Shumway (Figs. 192 and 193).

Fig. 189.—Dieffenbach's blepharoplasty.

Fig. 190.—The Dieffenbach-Szymanowski operation.

Blepharoplasty with a Pedicle from a Distant Part.—An ancient operation, which is known as the Italian method, is the application to the eyelids of a procedure which the Branca family and Tagliacozzi applied in the fifteenth and sixteenth centuries to the restoration of the nose. The flap is obtained from the inner aspect of the arm. Because of the necessity of prolonged immobilization of the parts the method has never become popular.

Blepharoplasty Without a Pedicle (Skin-grafting).—If the skin is obtained from the patient it is known as an autograft; if from another person, it is a heterograft; if from one of the lower animals, it is a

Fig. 191.—Knapp's blepharoplasty.

zoögraft. A heterograft or autograft generally grows better than a zoögraft, although the heterograft may be the means of transmitting syphilis.

Le Fort's Method (the transplantation of flaps comprising the whole thickness of the skin without the subcutaneous fat) has been practiced suc-

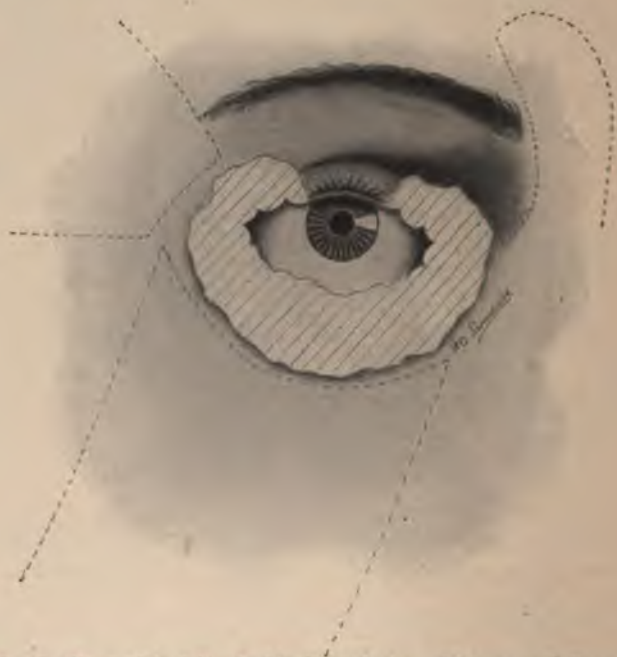


Fig. 192.—Outlines of incision in blepharoplasty. (Posey and Shumway.)

cessfully by le Fort, Sichel, Stellwag, and others. The procedure was popularized by the writings of Wolfe. In the operation from 30 to 50 per cent. should be allowed for shrinkage. Sutures may or may not be used. A dry dressing is applied.

REVERDIN'S METHOD.—In this procedure pieces of clean skin, one or two millimetres in diameter, are cut from the thigh or arm. The epithelium and a part of the corium are removed. Numerous such grafts are implanted upon the denuded surface at intervals of five to ten millimetres, and a gauze dressing is applied. This should not be disturbed for several days. If there is much suppuration the superficial layers of gauze can be changed daily and the parts can be washed with a solution of boric acid. Strong antiseptics should not be applied.

THIERSCH'S METHOD is generally believed to be the best means of closing skin-defects. Both the area to be filled and the surface from which

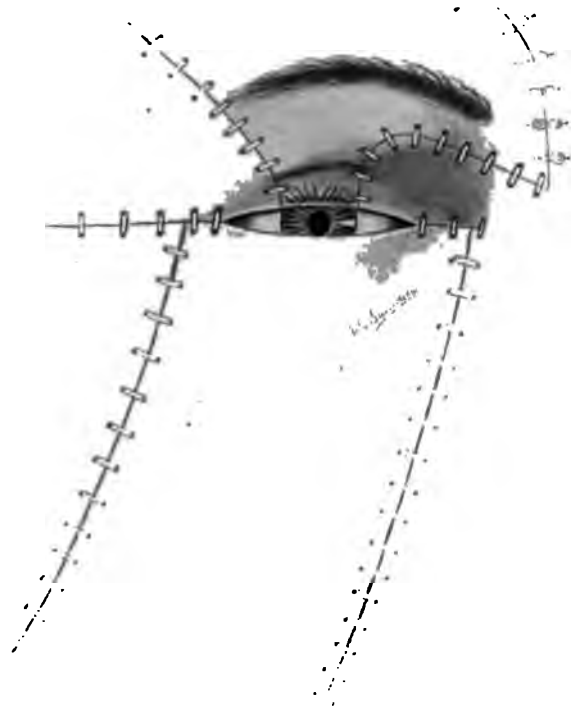


Fig. 193.—Result of blepharoplasty. (POSEY and SHUMWAY.)

grafts are to be cut must be clean (washed with soap and hot water and then with a solution of bichlorid of mercury) and should be kept moist with a physiologic salt solution (bicarbonate and chlorid of sodium, of each 0.3 per cent.). With a sterile razor strips are cut which are to include only the epidermal and Malpighian layers. In this procedure the skin is held tense and the strips are cut with a gentle to-and-fro movement. They are then floated in the normal salt solution on to the area which is to be covered. In this manner strip after strip is to be applied until the denuded area is covered. The strips should overlap one another and also should

cover the skin. All bleeding should be checked before the grafts are placed. During the operation there may be doubt as to which is the raw surface of a particular graft. This can be determined by remembering that the graft rolls up toward the raw surface. A layer of gauze or of rubber tissue is to be applied and held in place by a bandage. The dressing should be smeared with sterile vaselin, so that the grafts will not become detached when the dressing is changed. Thiersch grafts are not only of value in closing defects of the eyelids and face, but are also applied to the bones of the orbit in cases requiring total removal (exenteration) of the orbital contents. They are also of value in symblepharon operations. The grafts will grow upon periosteum, bone, tendons, fascia, and muscle.

CHAPTER VI.

DISEASES OF THE LACRIMAL APPARATUS.

SINCE the lacrimal apparatus naturally admits of division into a secretory and an excretory part, it will be proper to consider, first, the diseases of the gland, and, second, the diseases of the drainage system. Lacrimal diseases form about 2 per cent. of the practice of ophthalmologists.

DISEASES OF THE LACRIMAL GLAND.

The lacrimal gland of the orbit is subject to inflammation, injury, and new growths. The accessory glands also take on certain morbid actions.

Inflammation of the Lacrimal Gland (Dacryoadenitis) is a very rare disease, which may end in resolution, in suppuration, or in chronic adenitis. The inflammation may be acute or chronic.

ACUTE DACRYOADENITIS begins with pain, redness, and swelling of the upper lid and conjunctiva, particularly of the outer extremity of the lid. In the beginning of the attack an enlargement of the gland can be readily felt. On elevating the lid, the lower part of the swollen gland becomes visible. The skin is movable over the mass, and, in pronounced cases, the eye is protruded and depressed. The upper lid droops. In the acute stage the gland is very sensitive, and eversion of the lid cannot be performed. Under these circumstances the eye may present a picture resembling that of purulent conjunctivitis or orbital cellulitis. When suppuration occurs, the pus may escape through the skin of the lid or through the conjunctiva. Probably the lower anterior part of the gland, the so-called palpebral part, is more often inflamed than the main portion.

Etiology.—Dacryoadenitis is more common in women and children than in men and adults. It sometimes occurs in epidemics in connection with mumps. Injuries, exposure to cold, and certain blood diseases —such as syphilis, gout, rheumatism, scrofula, and sepsis—and direct infection from the conjunctival *cul-de-sac* are causes. Metastatic dacryoadenitis occurring in patients with gonorrhea has been described by Terson, Panas, and Ferry.

Diagnosis.—Acute dacryoadenitis must be distinguished from orbital cellulitis. The differentation can be made by the location of the point of greatest intensity of the inflammation. In some cases diagnosis may be impossible until after exploratory incision.

Treatment.—If a case of dacryoadenitis is seen in the earliest stage, the patient should be given a mild cathartic and any special medication which may be needed to combat any constitutional disease from which

he may suffer. The application of iced compresses is valuable. If the case is seen later, hot packs are to be applied to the region over the inflamed gland. Pus may form in a few days, in which case an incision must be made. The inflammatory symptoms may subside, and the case then becomes chronic.

Chronic Dacryoadenitis is recognized by the history and by the presence of a swollen, lobulated, tender mass, situated at the outer and upper angle of the orbit. In a case of this character, seen by Snell, there was almost a complete absence of tears. Chronic inflammation of the gland may appear at any age. It is found in non-syphilitics as well as in syphilitics. Often its cause cannot be determined.

The treatment comprises the local use of iodin and mercurial ointments, and the internal administration of iodid of potassium and bichlorid of mercury. There is abundant evidence of the efficiency of these remedies in those who do not give a specific history, as well as in syphilitics. Where there is a history of rheumatism, the iodid or salicylate of sodium is of value. The use of a compress bandage is recommended by Galezowski.

Dacryoadenalgia is the name applied by A. Schmidt to neuralgia of the lacrimal gland. The condition is an extremely rare one, and it is a question whether the term should be retained in ophthalmic literature. Possibly cases of supposed neuralgia of the gland were examples of lesions or functional disturbances located elsewhere.

Simple Hypertrophy, or Adenoma, of the Lacrimal Gland is rarely seen. It may be congenital or acquired. The enlargement may be so great as to cause optic-nerve atrophy by pressure and extrusion of the globe from the orbit. An interesting example of adenoma occurred in the practice of the late Dr. C. Johnston, of Baltimore. The eye, forced from its normal position, still retained its movements, and vision equal to the counting of fingers was preserved. On removal the gland was found to be the size of a hen's egg and contained numerous dacryoliths. The diagnosis of simple hypertrophy may be difficult at an early stage. A rapidly increasing growth may be a simple adenoma, a malignant neoplasm, or a tubercular tumor.

The treatment of hypertrophy should begin with the local use of iodin and the internal use of potassium iodid and bichlorid of mercury. Should this fail to check the process, recourse should be had to excision of the gland.

Atrophy of the Gland occurs rarely and is generally associated with xerosis of the conjunctiva. It may be caused by the pressure of an orbital tumor. Destruction of the lacrimal sac is sometimes followed by absence of lacrimation of the same side, presumably due to atrophy. There is no treatment for this condition.

Dacryoliths.—Concretions in the lacrimal gland are of rare occurrence. If recognized, they should be removed by incision.

Dacryops.—Cyst of the gland, or of an efferent duct, is a rare condition which may be present at birth, but is usually acquired. It forms a tumor varying in size from a pea to a pigeon's egg, situated at the upper and outer part of the upper lid, and extending backward to the orbit. On everting the lid, a translucent, elastic swelling, of a bluish-pink color, presents itself. If the patient weeps, it may increase rapidly in size. Pressure may cause it to collapse.

TREATMENT.—Dacryops is best treated by excision. Some authors have thought that, on account of the thinness of the wall of the cyst, excision is not possible. Mr. Arnold Lawson has reported a case in which this operation was successfully performed.

Hydatid Cyst of the lacrimal gland has been reported by Fromaget.

Fistula of the Lacrimal Gland may occur from trauma, dacryops, or abscess, or may be present as a congenital condition. The fistula opens on to the upper lid, and presents a minute orifice through which tears ooze forth. Under excitement or irritation the flow becomes profuse. The closure of such an opening is sometimes difficult, and, if the effort succeeds, it may cause dacryoadenitis. The older ophthalmologists resorted to heroic measures. Beer closed a fistula by passing a red-hot knitting-needle into the opening, and Mackenzie used a probe coated with lunar caustic. The simplest and most satisfactory way to deal with such cases is to excise the lacrimal gland, and at the same time cut out the tissue around the fistulous tract. Fistulæ due to caries or necrosis of the orbital wall will heal only after the removal of the diseased bone.

Dislocation of the Gland may be present at birth, follow trauma, or occur spontaneously. When it results from trauma, the parts should be cleansed, the gland replaced, the skin sutured, and a bandage applied. Bistis has recorded a case in which this treatment was successful. In traumatic luxation, where the gland is badly lacerated and the ducts are torn, it will be best to excise the mass. Its removal will not materially affect the moistening of the conjunctiva.

Spontaneous luxation rarely occurs, the gland commonly presenting above the outer part of the upper lid. Noyes found it beneath the ocular conjunctiva, over the insertion of the external rectus muscle. Robertson removed a lacrimal gland dislocated into the upper lid, where it occupied the whole length and part of the breadth of the eyelid. If luxated chiefly into the lid, the gland should be anchored to the periosteum of the orbit. If this fails, excision will be in order.

Syphilis of the Lacrimal Gland, although a rare condition, undoubtedly occurs. Such a case was recorded by Streatfeild. In any case of enlargement of the gland in which the cause is not known, antisyphilitic treatment should be instituted before surgical intervention is practiced.

Tumors of the Lacrimal Gland are not often seen. Aside from the cysts already mentioned, the new formations in this part are sarcomas of various kinds, carcinomas, myxomas, lymphadenomas, chloromas,

hydatids, dermoids, chondromas, angiomas, and concretions (dacryoliths). Buller and Byers have reported a case of primary carcinoma of the gland.

The malignant tumors here grow rapidly. In many cases it will be manifestly impossible to make a diagnosis of the nature of a tumor of the lacrimal gland until after removal. If the growth does not subside under antisyphilitic treatment, it should be excised.

Tuberculosis of the Gland has been described recently by de Lapersonne, L. Müller, Baas, Salzer, and Süsskind. The clinical features are the presence of a hard tumor, about the size of an almond, situated at the upper and outer part of the orbit. The tumor is movable and is not adherent to the skin. In one-half the cases the growth was rapid (two or three months) and suggested sarcoma. In the other half the disease progressed slowly (three or four years). In only one case was pain present at the beginning. In three cases the skin over the tumor was red and swollen. Nearly all the patients showed tuberculosis elsewhere. The movement of the eye was not limited. It is evident that the nature of the tumor in these cases can be determined only after removal. Microscopic examination shows typical miliary and submiliary tubercles, with more or less round-cell infiltration. The tubercle bacillus was found in less than half of the reported cases. Thus far caseation has not been reported. Surgical intervention will be in order only after medical and hygienic measures have failed.

Chloroma.—A rapidly growing and very malignant and rare tumor of the gland is chloroma, which Dock states "is a lymphomatous process similar in its clinical features to leukemia." The tumor is hard and becomes so large that the lids do not cover the eyeball. The lymphatic glands of the neck become enlarged, the blood shows an excess of leucocytes, and hemorrhages occur from the mucous membranes. In Ayres's case the immediate cause of death was hemorrhage from the conjunctiva. On removal the tumor presents a light-greenish color; hence the name. It is a disease of early life, the average age of the reported cases having been fifteen years.

Affections of the Accessory Glands are probably much more common than would be thought from the small amount of literature. Antonelli has described a case of dacryoadenitis of the inferior accessory lobules in a man aged twenty-five years. There was a small tumor, situated one centimetre up and out from the corneal limbus.

DISEASES OF THE DRAINAGE APPARATUS.

It is necessary to consider the affections of the puncta, canaliculi, sac, and duct. Diseases of these parts are characterized by an overflow of tears, a symptom which is known as epiphora.

The Puncta may be congenitally absent or closed. Multiple puncta are sometimes seen. As a result of trachoma, or from cicatricial contractions following burns and other injuries, or from ulceration, the puncta

may be misplaced. Drooping of the lower lid in facial paralysis causes the lower punctum to sag. Superficial occlusion of this also occurs in blepharitis marginalis.

The treatment consists in the introduction of a sharp-pointed lacrimal probe. If the canaliculus also is occluded, it will be necessary to probe it or to slit it up to its junction with the sac. In cases of inversion or eversion of the punctum it will be necessary to remove the cause (entropion, ectropion, inflammatory thickening of the lid).

The Canaliculi may be congenitally absent, in which case they are represented by furrows on the lid-margin, or occluded by cicatricial contraction, foreign bodies, fungous growths, or tumors. Steffan and others have met with a supernumerary canaliculus of the lower eyelid. In case the canaliculi are absent, an attempt should be made to secure an opening directly into the sac, as described under the head of lacrimal operations. In case a wound cuts a canaliculus across, the surgeon should attempt to pass a probe into the nasal duct and then insert a lacrimal style, which should be left *in situ* during the process of healing. Foreign bodies in the canaliculi are sometimes seen at the present time. The author has recently removed a birdshot from the superior canaliculus, where it had rested for nine years. Hairs, eyelashes, pieces of silk thread, and dacryoliths have been found in this part of the drainage apparatus. The author has met with one case in which the lower canaliculus was occupied by a growth of leptothrix, which formed a round, hard mass, producing epiphora and considerable inflammatory disturbance. The canaliculus, however, was not completely obstructed, since a stream of water from a syringe passed down the nasal duct.

Any foreign body, such as a cilium or a piece of the beard of barley or wheat, if projecting, can be seized with forceps and removed. If not projecting, the canal should be opened to a small extent and the offending substance should be picked out with forceps. Dacryoliths are to receive the same treatment. Polypi have been occasionally found in the canaliculi. Incision of the canal and removal comprise the proper treatment. Stricture of the canaliculus, particularly of the lower, is often found at the nasal extremity of the canal. Its treatment will be considered with that of stricture of the nasal duct.

Mucocele of the Inferior Canaliculus has been described by Brady, of San Francisco. The dilated canal measured eleven by six millimetres. It contained watery mucus, and was treated by destroying the lining epithelium with an electrocautery.

Leptothrix Lacrimalis (Tear-stone) is a fungus which is rarely found in the canaliculi. It is much more frequent in the lower than in the upper canaliculus. The inner part of the lid, corresponding to the canaliculus, is red and swollen, with a tendency to ectropion, the appearance resembling that of a stye. The disease is unilateral. According to Kipp, it occurs in the proportion of 1 to 53,600 eye cases. The

caruncle and plica semilunaris show a persistent conjunctivitis; and a tenacious, yellowish, creamy fluid exudes from the punctum. Noyes mentions a pumping or sucking movement in the dilated punctum. The fungous mass may be rounded and firm, feeling hard like a chalazion. The condition may be mistaken for hordeolum or for chronic dacryocystitis. From the latter it can easily be distinguished by the absence of distension of the lacrimal sac, and from the former by its chronic nature. Higgins met with a case of leptothrix which was mistaken for an epithelioma. Microscopic examination of the mass shows it to be composed of isolated, unbranched, straight, and very thin filamentous cells, similar to leptothrix buccalis found in the tartar of teeth. Leptothrix has the characteristic of secreting lime salts. When calcified, it forms a hard mass known as *tear-stone*. Up to 1874 the fungus found in these concretions was reported to have been leptothrix. Since that date streptothrix was found in all instances. Since 1894 actinomyces has been reported as present in such concretions. Evidently the subject demands further investigation. In the treatment of this disease pressure will cause the soft part to exude, but will not cure the condition. It will be necessary to slit the canaliculus and apply antiseptics to the portion distended by the mass. Here a mild solution of nitrate of silver will be useful.

Lacrimal Actinomycosis.—Schroeder, in 1894, in a concretion removed from a canaliculus, found microscopic appearances typical of the ray-fungus. He believes that all previous cases of obstruction of a canaliculus by fungus were due to the development of actinomyces. Elschnig and Evetsky agree in this opinion, which the author knows to be incorrect. Actinomycosis has been found a few times producing obstruction of the canaliculi. The treatment for actinomycosis includes slitting of the canaliculus, removal of the mass, curettement of the walls of the distended canal, and the application of a bichlorid solution (1 to 1000).

Inflammation of the Lacrimal Sac.—The lacrimal sac is subject to acute and chronic inflammation. The former is commonly known as abscess, the latter as blennorrhea of the sac. It would seem more rational to speak of inflammation of the sac as catarrhal or purulent.

Etiology of Lacrimal Inflammations.—In the vast majority of cases inflammations of the sac and duct are due to the condition of the nasal chambers. The extension of catarrhal inflammation into the nasal duct causes swelling of the lining membrane, with a damming up of tears. Heat, moisture, and stagnation furnish favorable conditions for the multiplication of germs, with which the lacrimal secretion is always well supplied. Mucus and pus form within the sac and duct, and, although this pus does not cause severe inflammation when brought in contact with the conjunctiva, it does produce disastrous results in corneal wounds or in ulcers of the cornea.

In a few cases periostitis with caries of the lacrimal bone, due to syphilis, produces a form of lacrimal stricture which is difficult to cure.

Struma, by acting on the nasal mucous membrane, must be regarded as an etiologic factor. A slight nasal catarrh may cause lacrimation, photophobia, and redness, in eyes which are otherwise normal, the excretory passages being permeable. In such cases spraying of the nose with a mild antiseptic and the instillation into the conjunctiva of a few drops of zinc-cocain solution (zinc sulphate, gr. ss; cocain, gr. j; water, ℥j) will be followed by relief. The next attack of rhinitis will reproduce the symptoms.

Certain cases of lacrimal obstruction are undoubtedly due to unskillful surgery. The habit, common among some practitioners, of slitting the canaliculus and passing probes upon every case showing epiphora, often results in the tearing of the membranous lining, the formation of cicatrices, and the production of infection. Such reckless treatment should be avoided. Occasionally a case of lacrimal obstruction is due to the lodgment of a piece of broken instrument, such as the point of a canaliculus knife, or the slipping of a style into the nasal duct.

As regards the nasal disease, a great variety is presented, embracing hypertrophic and atrophic rhinitis, rhinoscleroma, the growth of polypi, nasal tuberculosis, malformation of the inferior turbinated body, the growth of exostoses, the presence of neoplasms in the antrum of the superior maxilla, etc.

That a veritable trachomatous inflammation of the tear-sac exists has been demonstrated by Vincentiis, Cirincione, Kalt, Kuhnt, and Raehlmann. Wernke, who made sections of ten tear-sacs removed from trachomatous patients, found in them the same pathologic changes that exist in the conjunctiva in trachoma. This fact helps to explain the etiology of acute dacryocystitis. It is necessary to state, however, that the correctness of these observations has been disputed.

Lacrimal obstruction is common in the newborn. Stephenson found 1.75 per cent. among 1538 out-patients of a children's hospital. In many of these the history showed the presence of a discharge, either from or shortly after birth. The eye is generally not red, the cornea is clear, and it is exceptional to find a swelling behind the tendo oculi. A plug of mucus or muco-pus is present at the inner canthus. Pressure on the inner palpebral ligament causes a discharge to appear. That the etiology of this disease in the newborn is different from that of the adult is shown by the rarity of the evidence of syphilis, of nasal catarrh, or of bone disease. The cause is to be sought in the embryologic history of the sac and duct, obstruction in the newborn being due to delayed absorption of the material which exists during fetal life in the sac and duct, plus infection by bacteria. The bacteria found in the discharge from the sac and duct are numerous. They include pneumococcus, xerosis bacillus, staphylococcus pyogenes albus and aureus, bacillus coli communis, pneumobacillus, and bacillus fœtidus ozænæ.

Catarrhal Dacryocystitis.—This disease has generally passed the acute stage when the patient applies for treatment. In acute catarrhal dacryo-

cystitis the chief symptom is epiphora. The patient is usually suffering with an acute rhinitis, and the involvement of the sac is the result of direct extension of the inflammatory process from the nasal mucous membrane. The conjunctiva is hyperemic. The skin of the lids and that over the sac is often excoriated from scalding tears and from the use of the handkerchief. Pressure on the sac causes the appearance of a slight mucous discharge. The disease is commonly bilateral.

In cases of catarrhal inflammation which have become chronic there is often an accumulation of fluid in the sac, with a distension of its walls, forming above the tendo oculi a rounded, globular mass, known as *mucocele.* Pressure on the sac will cause the discharge of a thick, glairy mucus. While a mucocele is often small, it may attain enormous dimensions, as in a case recorded by Kenneth Scott, in which the distended sac reached from the inner angle of the eye to the angle of the mouth.

TREATMENT.—In acute catarrhal dacryocystitis it will be necessary to treat the nose. The use of soothing sprays to the nasal mucous membrane will be beneficial. If there is great swelling of the membrane the application of suprarenal extract or of adrenalin solution will serve to reopen the nasal passage. This should be followed by the use of an oleaginous spray, either with or without menthol.

The nasal treatment is to be followed by irrigation of the sac. For this purpose the lower punctum can be rapidly dilated to a size sufficient to admit the nozzle of the irrigator and an ounce or two of warm boric acid solution is caused to flow through the excretory channels. The application of very hot compresses to the region over the sac will be grateful to the patient and beneficial to the inflammation. In this condition cutting of the canaliculus is absolutely contra-indicated. The prognosis is favorable only when it is possible to treat the nasal chambers carefully and persistently. After the acute symptoms have subsided, the state of the refraction should be determined.

In cases among the newborn pressure is to be applied daily over the sac, for the purpose of removing the secretion and opening the naso-lacrimal duct. Under this simple treatment most cases improve rapidly. Where this is insufficient, the surgeon should dilate the lower canaliculus and pass a probe, under general anesthesia. The canaliculus should not be cut.

Suppurative Dacryocystitis.—As the result of an acute attack of inflammation not ending in resolution, or from repeated attacks of catarrhal inflammation, the sac becomes filled with a purulent collection (Fig. 1, Plate VIII). The cause of most cases can be found to be a stricture of the naso-lacrimal duct. Suppurative dacryocystitis appears in two forms: the acute and the chronic.

ACUTE SUPPURATIVE INFLAMMATION OF THE SAC begins often with a chill. There is pain at the inner canthus. In the region of the sac a hard, brawny swelling appears, which closes the eyelids and often is

PLATE VIII.

External Diseases of the Eye.

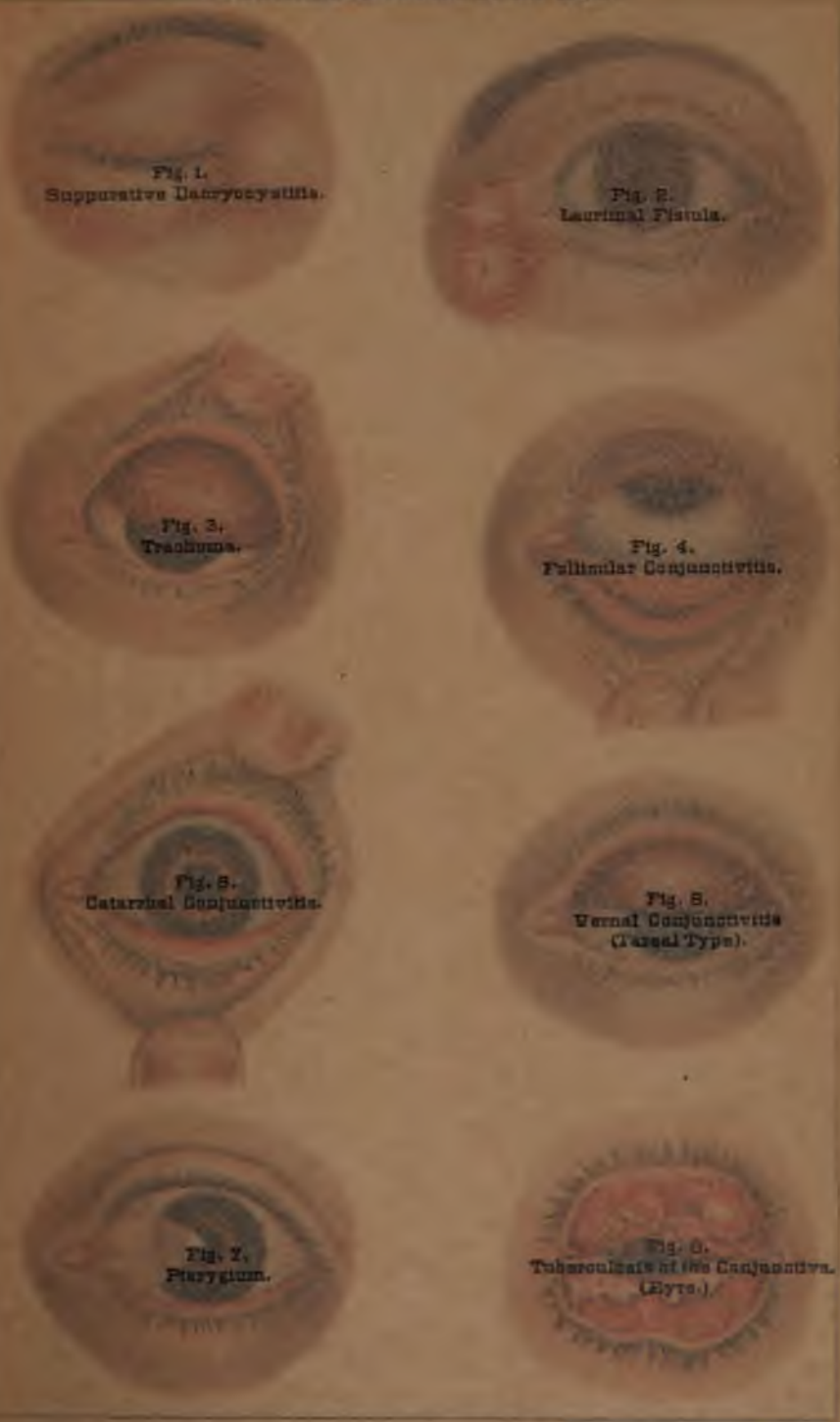


Fig. 1.
Suppurative Dacryocystitis.

Fig. 2.
Lacrimal Fistula.

Fig. 3.
Trachoma.

Fig. 4.
Follicular Conjunctivitis.

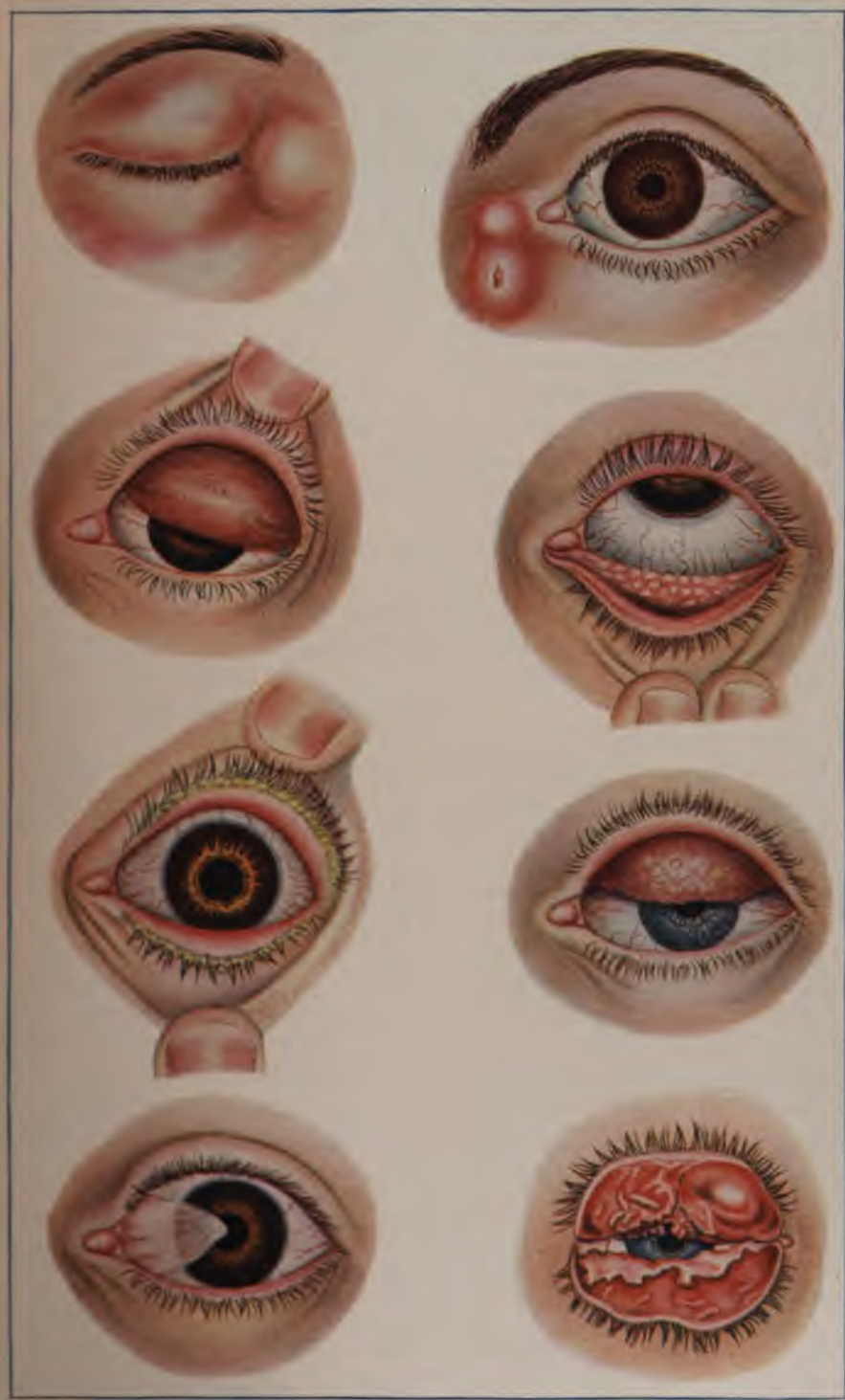
Fig. 5.
Catarrhal Conjunctivitis.

Fig. 6.
Vernal Conjunctivitis
(Tarsal Type).

Fig. 7.
Pterygium.

Fig. 8.
Tuberculosis of the Conjunctiva.
(Elyot.)

PLATE 8



so extensive as to resemble orbital cellulitis. The greatest redness, however, is found in the inner extremity of the swelling. The pain, caused by distension of the sac, is severe. After several days the swelling becomes softer and diminishes in size. The inflammatory symptoms may gradually subside and may leave a chronic suppurative inflammation; or, as often happens, the abscess bursts through the sac and skin, thus forming a *fistula* (Fig. 2, Plate VIII). Repeated attacks of acute inflammation occur until after the stricture has been opened and treated.

Treatment.—In the beginning of an attack of acute inflammation of the sac the use of cool compresses will be beneficial. A saline cathartic should be given and the patient should be placed in bed. After the hard, brawny swelling has appeared, hot applications are preferable. Often the pain is so severe that anodynes are necessary. When the swelling begins to fluctuate, under an anesthetic the surgeon should slit the upper canaliculus and pass a series of probes through the stricture, dilating it *ad maximum*. The after-treatment consists in daily irrigation with an antiseptic solution (pyoktanin, 1 to 500; creolin, 1 to 20; silver nitrate, 1 to 300) and probing until the discharge begins to lessen, after which the probing can be repeated at intervals of two or three days until recovery takes place. In these cases it will be necessary to use the largest probes the canal will accept. As regards irrigation, astringents of many kinds have been tried. The hot-water treatment, combined with probing, is probably as efficient as any method.

Chronic Suppurative Inflammation of the Sac causes epiphora and often produces an obstinate conjunctivitis. Pressure on the sac leads to a discharge of pus through the canaliculi. This disease is caused usually by stricture of the nasal duct. The proper treatment is to dilate the stricture and use irrigation.

Obstruction and Stricture of the Lacrimo-nasal Duct.—It is necessary to differentiate between obstruction and stricture. In the former, there is swelling and hypertrophy of the lining membrane of the duct, caused generally by the upward extension of the nasal disease (a swollen fold of the sac obstructing the lumen), or by the lodgment of a foreign body from above, or by eyestrain. Extension from below is common in exanthematous diseases, because of the accompanying inflammation of the naso-pharyngeal mucous membrane. It is also found in nasal polypi, and in hypertrophy of the inferior turbinated body. In these cases the change is limited to the lining membrane of the duct. The lodgment of cilia and other foreign bodies is probably more common than has been supposed. True anatomic stricture is rare. Its causes are predisposing and exciting. Among the former must be classed the various malformations of the duct; and among the latter suppurative inflammation of the sac, the production of false passages by unskillful attempts at probing, and the use of caustics. True stricture is occasionally due to an exostosis within the duct, the growth of a neoplasm situated within the

nose or springing from the lining membrane of the antrum of Highmore, or the fracture of bones forming the duct. The cicatrization of a syphilitic or tuberculous ulcer, and the contraction attending atrophic rhinitis, are also causes of true stricture. In a few instances the whole lining membrane of the duct is converted into a fibrous cord.

Symptoms.—The symptoms of obstruction and stricture are often masked. Epiphora may be the only external sign of disease. The presence of mucocele or of blennorrhea of the sac generally indicates stricture, but may be due to simple obstruction.

Diagnosis.—In making a diagnosis between simple obstruction and true stricture it will be necessary to employ the lacrimal syringe and probe and to take into consideration the condition of the nose. The surgeon should inject a few drops of a colored solution into the lower canaliculus, while the upper one is compressed. If the fluid passes into the nose, the duct is open. If it returns, there is either obstruction or true stricture. The diagnosis between these will require the passage of a probe through the rapidly dilated canaliculus. After enlarging the canaliculus, preferably the upper one, and passing the probe well into the sac, the syringe should be used again. If the solution now passes into the nose, the conclusion is that the obstruction has been overcome and the deeper use of probes will not be necessary. If the passage is not free, the probe must be used in the duct proper, and the obstruction or stricture located. The injection of a solution of the extract of the suprarenal gland into the duct will cause an obstruction to disappear, while having little or no effect on true stricture.

Prognosis.—The prognosis of chronic affections of the duct must be guarded. Many of these cases will improve while under treatment, but will relapse.

Treatment.—In all cases of obstruction to the passage of tears it will be advisable to begin with the examination and treatment of the nasal mucous membrane. The use of cocain or suprarenal extract will overcome swelling and enable the surgeon to inspect the condition of the inferior turbinated body and adjacent parts. Polypi should be removed; hypertrophies should be treated with the galvanocautery; and atrophic rhinitis will call for frequent cleansing of the passages. Simple rhinitis will improve under the use of Dobell's solution and the application of a spray of menthol in albolene. Adenoids should be removed and the condition of the whole naso-pharyngeal tract must be improved.

After this has been done the sac and duct should be washed out daily with hot boric-acid solution. Errors of refraction should be corrected and the general condition of the patient should be looked after.

Under this conservative treatment many cases will either result in a cure or their condition will be so much relieved that more radical treatment will not be required. There will remain, however, a certain percentage of cases in which, in addition to the daily washing of the duct,

it will be necessary to pass probes at intervals of three or four days. The probing should be done through the dilated and uncut canaliculus. By patience, the surgeon will generally succeed in passing the probe without cutting the canaliculus. If there are cicatricial bands, it will be safer to cut them with a curved canaliculus knife (passed through the dilated canaliculus) than to risk the formation of a false passage by the use of the probe.

Immediately after cutting the resisting bands the probe is to be passed. When the obstruction is located at the junction of the sac and duct, or is in any part of the upper half of the duct, the probe of Snellen (Fig. 197) is preferred. This instrument has a bulbous tip and is made on the principle of the wedge, thus giving the greatest dilation at the place where contraction is often found, viz.: the junction of the sac and duct. The probe should remain in position for ten minutes, when it is to be withdrawn and the duct is to be washed with boric-acid solution. The manipulation necessary to cut the fibrous bands, without cutting the canaliculus, will be explained in the latter part of this chapter.

In case a true stricture exists, the treatment mentioned above will show its site and resistance. If an exostosis is found in the lower part of the duct, an attempt should be made to tap the middle nasal meatus, as is explained under lacrimal operations. Such a condition, however, is rare, and generally treatment gives poor results. In ordinary cases of stricture some force is required to pass the probe, but it must be intelligent force; and no surgeon should operate on the living who has not repeatedly practiced on the dead. It may happen that the stricture is located at the lower end of the duct; if so, the use of (Theobald's) long probes will be necessary, and the canaliculus should first be cut. Whenever cutting of the canaliculus is required, it will be best to incise it in only part of its extent. Often a cut one-sixth of an inch in length will suffice. Such a short incision, while permitting the use of the probe, will not entirely destroy the function of the canal.

Cases occur in which, although probing and irrigation are easily accomplished, troublesome epiphora persists. In this event search should be made for pathologic conditions within the nose, causing a valve-like fold of mucous membrane to occlude the duct.

In the treatment of lacrimal stricture by probes only the metal instruments are now used. The method of Gensoul, who passed the probes from the nasal end of the duct, is not now in use. It is possible, however, to probe the lower half of the canal by this method, and cases may be seen in which this procedure will be of value. In operating from above, it may happen that the surgeon will be in doubt whether the probe has passed into the nasal fossa. In such an event, the nasal speculum should be introduced, the parts should be illuminated, and another probe should be passed between the inferior turbinated bone and the outer nasal wall until it touches the instrument in the lacrimal duct.

In cases of true stricture which do not yield to the plan of treatment here outlined, the surgeon can resort to one of five procedures: 1. The canaliculus can be slit in its entire length and probes of large calibre used. 2. A style can be introduced and worn for several months, with the expectation of producing absorption of the stricture walls. 3. The surgeon can excise the walls of the stricture by chiseling away the anterior osseous wall, after which a flexible catheter can be left in the duct until cicatrization is complete. 4. The stricture may be treated by electrolysis. 5. The lacrimal sac and gland can be excised.

1. Although the first method has the sanction of some eminent names, it is not to be looked on with favor. The forcing of large probes through the lacrimo-nasal duct often gives immediate improvement, which, after a few months, is followed by the formation of a stricture at the point where the canaliculus joins the duct; or the lining membrane of the duct may be converted into a firm cord of fibrous tissue, extending from the sac to the nasal chamber. This unfortunate result frequently occurs. If the canal remains open, the patient finds that, whenever he blows his nose, air and mucus are forced upward into the conjunctiva, and thus the eye is exposed to infection from this source.

2. The introduction and wearing of a style is a very old method of treatment and sometimes is an efficient one. Numerous substances have been used for this purpose. Probably a piece of large silver wire is as good as any. Hollow styles are also in use. An important point is to provide the style with a flange which shall be long enough to prevent the instrument slipping down into the duct. If this accident occurs, and efforts at removal are unsuccessful, a silver style can be destroyed by the corroding effect of weak solutions of iodin injected repeatedly into the duct. The style can be removed from time to time and the duct examined to determine whether its lumen is increasing. Generally a style must be worn for several months. Weidler has recorded a case in which orbital cellulitis followed slitting of the canaliculus and the use of a leaden style.

3. Excision of the walls of the stricture, particularly when the obstruction is osseous, is a method of treatment which has occurred to the author as having a possible value in a few rare cases which do not yield to less heroic methods. After removal of all offending tissue, the operation having been made under aseptic precautions, it will be advisable to place a style or possibly a piece of rubber catheter in the duct, removing and reintroducing it from time to time, until cicatrization is complete.

4. Electrolysis, as a method of treatment of stricture of the lacrimal passages, was originally proposed and used by Tripier and Desmarres about thirty years ago. The latter pronounced against the method, which was afterward tried by many ophthalmologists and abandoned by most of them. Recently Lagrange, Chabaneix, and Fournier have made a complete study of the subject. They regard this form of treatment as valuable. Lagrange believes that electrolysis will effect a cure of lacrimal

stricture rapidly, harmlessly, and painlessly. That part of the probe lying in the canaliculus must be insulated; otherwise this portion of the excretory apparatus will receive the full force of the electrolytic action, and obliteration of the canaliculus will result. The current must be a weak one, insufficient to produce a caustic effect on the mucous lining of the nasal duct. The technique of the procedure will be discussed in the latter part of this chapter. Mial has recently pronounced in favor of electrolysis, which, he says, opens the lacrimal passages "speedily, painlessly, and without hemorrhage, and, furthermore, is antiseptic in its action."

5. Excision of the sac and gland can be practiced, as will be explained in the latter part of this chapter.

Fistula of the Lacrimal Sac, which has rarely been seen as a congenital condition, generally follows the breaking of an abscess, which opens below the tendo oculi (Fig. 2, Plate VIII). The fistula may open directly into the sac or may run a tortuous course. The edges of the opening are often hard, thickened, and ulcerated, and present exuberant granulations, and a discharge of tears and muco-pus. In some cases caries of the bone is present. The presence of a fistula in this location points to the existence of a stricture, which must be treated. Fistula is artificially produced when the surgeon incises the anterior wall of the sac to evacuate pus in suppurative dacryocystitis. In treating fistula it will be necessary to slit the canaliculus and pass the probes through the stricture. This treatment, combined with antiseptic injections, will cure nearly all cases. If, after the patency of the duct has been restored, the fistula refuses to close, a red-hot wire can be passed into it. The closure of the fistula is not to be attempted until after the stricture of the nasal duct has been removed. In these cases the presence of the fistulous tract is a safeguard against another attack of acute dacryocystitis.

LACRIMAL OPERATIONS.

I. Operations on the Lacrimal Gland are divisible into (1) those on the orbital lacrimal gland, and (2) operations on the palpebral gland.

1. EXTIRPATION OF THE ORBITAL PORTION is indicated both in health and in disease. According to C. R. Holmes, the normal gland and diseased sac are to be removed:—

1. In troublesome and otherwise incurable epiphora.
2. In cases where operations involving the opening of the globe must be performed and there is not sufficient time to apply other treatment.
3. In patients who lack the time for, or are unable to endure, probing.

The diseased gland should be removed:—

1. In adenoma which does not respond to other treatment.
2. In adenosarcoma and carcinoma.

Technique.—The field of operation having been shaved and made surgically clean, under general anesthesia a curved incision, four centimetres in length, is made along the base of the orbit over the site of the gland. For cosmetic reasons the cut should be made in the field of the eyebrow. The lower end of the incision should reach to the insertion of the outer canthal ligament. The tissues are separated down to the bone, the wound being opened by retractors. The connective tissue is incised over the gland, which is brought forward by forceps and shelled out of its bed. In operating upon a diseased gland the surgeon should be certain to remove all indurated, swollen, and diseased tissue. The lips of the wound are sutured and a dry gauze dressing and bandage are applied. Healing is uneventful.

Fig. 194.—Probes passed into the canaliculi to show their direction. (Author.)

(Original drawing by Dr. R. W. Mills.)

2. Excision of the Palpebral Gland is indicated in troublesome epiphora, not curable by other means, and in neoplasms. A local anesthetic is used. The surgeon seizes the everted upper lid, draws it upward and away from the globe, and makes an incision through the conjunctiva. The gland is held with forceps and excised. Owing to the glandular character of the mass, removal of all parts of it is difficult. The wound need not be stitched. This operation cuts the efferent ducts of the orbital lacrimal gland.

Obliteration of the Excretory Ducts by the galvanocautery is practiced by Bettremieux. Repeated cauterizations are necessary.

The Effect of These Operations is to diminish epiphora when the normal tissues are removed, and to remove deformity and exophthalmos when the diseased orbital gland is excised. Except in the presence of atrophy, the conjunctiva does not become dry as a result of these procedures, for

the reason that a sufficient amount of lacrimal fluid is secreted by the accessory glands, which are scattered through the lids.

II. Operations on the Excretory Apparatus.—These are the following :—

1. Opening of a closed punctum.
2. Syringing of the excretory apparatus.
3. Dilation of the canaliculus.
4. Cutting of the canaliculus.
5. Excision of the lacrimal sac.
6. Opening of naso-lacrimal stricture.
7. Opening of an obliterated nasal canal.
8. Insertion of a lacrimal style.
9. Obliteration of part of the apparatus.
10. Electrolytic treatment of stricture.
11. Incision of the anterior wall of the lacrimal sac.

1. The Punctum can be Opened by drawing the lid outward from the globe and steadying it by traction made toward the outer canthus. Then a needle or conical probe is passed perpendicularly to the lid-margin for one millimetre; it is then turned to a horizontal direction, and passed into the canaliculus. The natural direction of the canals is shown in Fig. 194. This procedure is necessary as a preliminary step to syringing the duct and to the passage of probes without slitting the canaliculus.

Fig. 195.—Lacrimal syringe. (McFarlan.)

2. Syringing the Excretory Apparatus is accomplished by passing the tip of Anel's, Meyer's, or McFarlan's instrument into the punctum and canaliculus. The fluid is then discharged and the effect is noted. If the fluid returns, it is evidence of a stricture in some part of the excretory apparatus. If the fluid passes into the nose, the canals are pervious. If the fluid passes downward into the naso-lacrimal duct, care should be taken not to tear the lining membrane. Syringing is valuable (1) as a diagnostic measure and (2) for therapeutic reasons. In the latter instance lavage of the duct is of great value in the treatment of chronic purulent inflammation. The apparatus which is shown in Fig. 196 is of value. It consists of a reservoir, a rubber tube, and a probe-pointed cannula.

3. Dilation of the Canaliculus is necessary whenever the surgeon wishes to pass a probe into the duct without cutting the canaliculus. The latter operation is resorted to in many cases where the former would suffice. The canaliculus can be dilated easily by passing probes of increasing size. Often an annoying epiphora will disappear after this little operation.

4. Cutting of the Canaliculus is necessary in the removal of foreign bodies, fungous growths, or calcareous masses; in eversions of the punctum causing epiphora; as preparatory to syringing the deeper parts of the excretory apparatus; and in case the passage of large probes is required. It is indicated in cases where the discharge from the sac is distinctly purulent, where the parts around the sac are red and tender, and where an abscess or fistula exists.

The operation requires the probe-pointed canaliculus knife of Weber or that of Stilling. The surgeon, standing behind the patient and supporting his head, passes the beak of the knife into the punctum. The lid is stretched and steadied by traction made on the outer canthus. The knife, with edge turned slightly toward the globe, is then pushed into the canaliculus and is made to pass from the horizontal to the vertical position. This movement cuts the wall of the canal. There is slight bleeding, and

Fig. 196.—Apparatus for lavage of the naso-lacrimal duct. (Author.)

no dressing is necessary. The wound must be kept open by daily probing, as otherwise it will heal.

5. Excision of the Lacrimal Sac has been done for these conditions: chronic dacryocystoblennorrhea, chronic stricture with discharge, occlusion of the nasal canal, chronic lacrimal fistula, and large mucocele. In addition to these, removal of the sac may be advisable where, in chronic inflammation, the patient's circumstances do not permit the long course of treatment needed to effect a cure, and in cases in which an operation requiring the opening of the eyeball is imperative. Excision of the sac should not be undertaken lightly.

Technique.—The field of operation and adjacent parts having been made surgically clean, the nasal cavity is plugged with gauze to prevent the flow of blood into the pharynx. The sac, duct, and nose should have been previously irrigated. A thick solution of starch, colored with iodin, is injected into the sac to render its outlines clear (C. R. Holmes). In

order to prevent infection, some operators obliterate the canaliculi by the galvanic cautery. This procedure also prevents the establishment of a fistula. An incision is made, beginning at a point just below the lower border of the middle of the tendo oculi and extending one inch downward and outward. There is likely to be free hemorrhage in this operation and it will be necessary to clamp several small arteries. The skin is now dissected carefully from the sac, which, if possible, is to be excised unopened. The lips of the wound are held apart, and, by means of scissors and a blunt chisel, the sac is separated from its bony bed. It is then excised close to the nasal duct and the duct and bed of the sac are thoroughly curetted. The surgeon should be sure to remove all parts of the sac, as otherwise the suppuration will return. Great care is necessary to prevent inundation of the wound and globe with the contents of the sac. Such material is highly infective, and, in the presence of a corneal wound or ulcer, may cause disastrous inflammation. The wound is to be closed with catgut sutures. A compress bandage with heavy tampons is applied over the operated area.

6. CATHETERISM (PROBING) OF THE NASAL DUCT is necessary in stricture. It is a painful operation and one that requires great skill. It

Fig. 197.—Lacrimal probe. (SNELLEN.)

is perhaps necessary to say that the beginner should practice passing probes on the cadaver before employing them on the living. The usual direction of the nasal duct is downward, backward, and outward, but the variations are many. The choice of a probe is important. The old Bowman instrument should never be used. Only those probes bearing an olive-shaped tip are to be employed. Of these the author prefers that of Snellen (Fig. 197). It combines the proper tip with the wedge shape, and thus possesses two advantages; the tip cannot well make a false passage, and the greatest dilation is at the junction of the sac and duct —a point where stricture is often located. After using Snellen's probes a few times, the instruments of Theobald can be employed. Their chief advantages lie in their length and in the gradual increase in size. The author believes in the use of the largest probes compatible with the integrity of the tissues, but he does not believe in the forcing of large probes into the duct.

Technique.—The canaliculus having been widely dilated or cut, and the sac emptied by pressure, the surgeon, standing behind the patient, passes the probe horizontally into the canaliculus until the lacrimal bone is felt. The probe is then to be withdrawn one millimetre and placed vertically; then it is passed downward into the duct. Gentle pressure is

used, and, if an obstruction is met, the probe is withdrawn, its curve is altered, and another attempt is made to pass it. At the lower end of the duct an obstruction is often felt, which means that the probe is impinging on the osseous wall of the canal. In such a case the probe is to be withdrawn slightly, and its direction slightly changed. Up to this point the concavity of the probe has faced outward, but to pass it into the nasal meatus it will be necessary to turn it until the concavity faces directly forward, or even forward and toward the opposite side. The complete passage of the probe will be evidenced by the removal of the impediment and by the fact that it passes from five to ten millimetres farther. The patient will complain of pain when the tip of the instrument strikes the lining membrane of the floor of the nasal meatus.

Many impediments may be met. The most frequent one arises from the point of the probe becoming entangled in a fold of mucous membrane of the sac at the time the instrument is moved from the horizontal to the vertical position. To use force under such circumstances is to cause a false passage. When such resistance is met, the probe should be withdrawn to a slight extent and another attempt should be made to enter it. The tearing of the lining membrane of the duct is not such a trifling accident as might be supposed, since it not only may cause irremediable stricture, but it opens the door to infection and in rare cases has caused phlegmonous inflammation, orbital cellulitis, loss of vision from optic-nerve atrophy, and even meningitis. The probe may slide readily over the first obstruction, but its point impinges on the lateral wall of the duct. In such a case the curve of the instrument must be changed or its direction altered. If, after two or three attempts, it fails to pass, the best procedure is not to continue farther at this sitting, but to dilate *ad maximum* the part of the duct which has been opened. This is done by rapidly increasing the size of the probe, which is to be left *in situ* for from fifteen to thirty minutes. A few days later an attempt can be made to finish the work.

A method of treating lacrimal stricture which aims to preserve the canaliculus intact has been devised by Caldwell, who, after passing a small probe through the upper canaliculus, stretches the skin at the outer canthus so as to render the point of the probe prominent. The surgeon then cuts down upon the probe and into the sac. A suture is then taken from the inner lip of the wound, including the sac, and left long for future use. Through this incision probes can be passed, the duct can be irrigated, and the site of the stricture is immediately located. When found, the stricture is cut with the probe-pointed canaliculus knife. Then the dilation is continued up to a No. 12 probe, if the duct will admit it. Closure of the wound in the sac can be prevented by the introduction of a piece of rubber drainage tube, until such time as the obstruction in the duct has been overcome. Then the lips of the wound are freshened and united with catgut sutures. Caldwell's probe (Fig. 198) consists of a staff, 11

centimetres long, with a bullet-shaped tip, 1 centimetre long, on each end. The tips are graded in size, each unit representing 2.5 millimetres of diameter.

7. Opening an Obliterated Nasal Canal can be attempted in those cases in which a bony stricture exists, due to exostosis, fracture, or caries. It is also indicated in those firm, fibrous strictures which have resisted ordinary methods of treatment. In two cases of bony closure, operated on by Caldwell, the nasal duct was successfully tapped in the middle nasal meatus, by means of an electric burr, after passing the probe into the duct as far as the site of the stricture. The operation requires careful measurements and exact anatomic knowledge. If this procedure fails, the surgeon can obliterate or excise the sac and remove the lacrimal gland; or the excision of the stricture can be attempted. The latter will require general anesthesia, and a scar will be left on the face. The space beneath the inferior turbinated bone, and that between the middle and inferior turbinated, is to be packed with absorbent cotton or gauze, to prevent the flow of blood into the pharynx. After passing a probe down to the point of stricture, the surgeon incises the soft parts, turns them back, separates the periosteum from the bone overlying the stricture, and

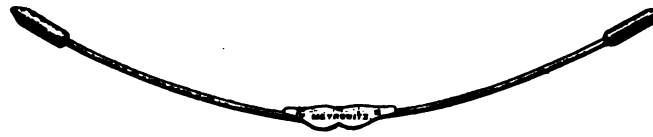


Fig. 198.—Lacrimal probe. (Caldwell.)

then chisels away the osseous wall. The exostosis is removed, and a small style is passed through the naso-lacrimal duct to a point below the site of the stricture. The soft parts are then united by fine catgut sutures.

8. Insertion of the Lacrimal Style.—The style is of use chiefly in those patients who live far from a surgeon or who can return for treatment only at long intervals. Gold, silver, lead, platinum, and rubber are among the materials most frequently used. An excellent style is one made of No. 25 platinum wire. The tubular styles are useless, becoming clogged with dust and mucus. Guiata employs the decalcified bone of a turtle's thigh as a style, and reports many cases cured. Whatever the material used, the style is to be provided with an elbow sufficiently long to prevent the instrument from slipping into the duct. The lower canaliculus having been cut and the duct having been dilated by probes, the style is given the same curve as that of the probe, and is introduced. When properly inserted, the instrument cannot be seen. Slight discomfort is caused for a few hours by its presence, but soon the patient ceases to note this. It should be removed monthly and the surgeon should observe the effect produced. Absorption of the stricture walls generally is complete in three or four months. The instrument can then be discarded. In many cases, however, the epiphora returns.

9. Obliteration of Part of the Lacrimal Apparatus.—The puncta and canaliculi may be obliterated by the use of a galvanocautery or by a ligature. The operation is performed for the purpose of preventing secretion from an inflamed lacrimal sac reaching the globe and thus possibly causing infection in accidental injuries of the cornea and in operations requiring opening of the globe. Obliteration of any part of the lacrimal apparatus is best done by means of the galvanocautery.

10. Electrolytic Treatment of Stricture.—In the use of electrolysis Lagrange employs from 2 to 4 milliampères for two or three minutes, the *séances* being repeated every third or fourth day. A rheostat is used to prevent too high a degree of electrolytic action. The sound is to be insulated for a short distance, corresponding to the extent of the canaliculus. This constitutes the negative pole; the positive pole can be placed on the nape of the neck, or, as Gorecki advises, it can be introduced into the nostril of the same side. This treatment is to be used in conjunction with probing.

11. Incision of the Anterior Wall of the Lacrimal Sac is a simple operation which is best done with a Beer knife, and does not require detailed description. The operation is indicated in phlegmonous inflammation, in mucocele, and as a prophylactic measure in corneal ulcers, injuries, or operations where chronic dacryocystitis exists. Extensive opening of the sac is necessary in the removal of foreign bodies and as a step in the operation of obliteration.

CHAPTER VII.

DISEASES OF THE CONJUNCTIVA.

THE conjunctiva is liable to congenital anomalies, tumors, inflammations, hypertrophies, degenerations, atrophy, tubercular and leprous infections, and injuries. These affections will be considered in the order mentioned. The conjunctiva is often involved in the cutaneous diseases of the lids; and, from its direct connection with the nasal mucous membrane via the lacrimo-nasal duct, it is influenced by morbid conditions of the nose and throat. Pathogenic microörganisms can be carried to the conjunctiva in many other ways: by the hands, towels, handkerchiefs, by water in swimming-pools, by dust, and by flies and other insects. Normally the conjunctiva always contains bacteria, twenty-eight varieties having been isolated by Eyre. Of these the staphylococcus epidermidis albus, as Randolph has shown, is found so frequently that it must be regarded as a regular inhabitant of the conjunctiva. Fortunately this coccus possesses but slight pathogenic properties. It is probable that bacteria which are non-pathogenic in the normal conjunctiva may become harmful when this membrane is injured or irritated. The temperature of the conjunctiva, which is normally 96° F. (Silex), is increased in disease. On account of the source of its blood-supply, the conjunctiva is involved to a certain extent in inflammation of the cornea, iris, and ciliary body. Conjunctival diseases are common, forming about 30 per cent. of ophthalmic cases in this country. Their frequency varies much with climate, latitude, and race, and may range from 10 to 90 per cent.

CONGENITAL ANOMALIES.

A few cases of congenital thickening of the conjunctiva, resembling pterygia and extending between the fissures of the lid, have been observed. The dermoid tumor, which is congenital, will be considered elsewhere. In cryptophthalmos the conjunctival sac is absent.

TUMORS OF THE CONJUNCTIVA.

Benign and malignant growths appear in the conjunctiva. Among the former are dermoids, lipomata, fibromata, polypi and granulation tumors, vascular tumors, osteomata, cysticerci, and lymphectasiæ. The malignant growths are sarcomata and epitheliomata. The diagnosis of the nature of a conjunctival tumor may be difficult or impossible until after a microscopic examination.

BENIGN TUMORS.

Dermoid Tumor.—The dermoid tumor, which is a congenital growth, generally is a flat, solid mass of a reddish or whitish color. It is situated partly in the conjunctiva, partly in the cornea, and is anchored to the latter. Often the surface is dry and is covered with downy hairs. Frequently other congenital anomalies are present. Demours met with dermoid located on the caruncle, in which the same eye showed an eccentric pupil. Wardrop described a dermoid of the conjunctiva from which twelve long hairs grew and hung over the cheek, as in the case shown in Fig. 199. In Wardrop's case the hairs did not appear until the sixteenth year, when the beard began to grow. The tumor is practically an island of skin planted on the eyeball. Histologically it presents a connective-tissue stroma, covered with epidermis and containing hair-follicles, sweat-glands, and sebaceous glands (Fig. 200). If thoroughly removed it does not return.

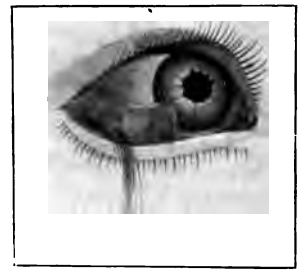


Fig. 199.—Dermoid tumor of the conjunctiva.
(Von Ammon.)

Subconjunctival Lipoma.—Another congenital growth is the lipoma, which may be single or multiple. This tumor is often quiescent until the time of puberty, when it begins to grow. It is covered by conjunctiva, is movable upon the eyeball, presents a yellowish color, and generally forms only one tumor-mass, but exceptionally is lobulated. It may become two centimetres in breadth, but is generally half this size. Sometimes lipomata arise from the orbital fat and cause the conjunctiva to bulge near the lacrimal gland and between the insertions of the recti muscles. Usually this is the only growth present, but Fuchs shows an illustration of a subconjunctival lipoma and a corneal dermoid in the same eye of a girl aged thirteen years; and Burnett, under the name dermolipomata, describes tumors containing both fat and skin elements. These tumors do not return after removal.

Fibroma is rarely found in the conjunctiva, where it forms a broad, flat growth occupying usually the upper *cul-de-sac*. Microscopically it is composed of interlacing strands of fibrous connective tissue with few vessels and with practically no interfibrillary substance. These tumors

do not recur. They are supposed to begin as polypoid growths which in time are changed into simple connective-tissue masses.

Myxoma has been observed very rarely in the conjunctiva.

Polypi, Papillomata, and Granulation Tumors can be described together. *Polypi* are soft or semisolid pedunculated tumors, covered by mucous membrane, and generally spring from the fornix. Usually multiple, small, and hidden, they may grow and project between the lids. They may become ulcerated. They are usually caused by the irritation of a retained foreign body. When this is removed and the polypus is snipped off, a cure readily follows. Histologically they are small fibromata pushing the conjunctiva in front of them. *Papillomata* spring

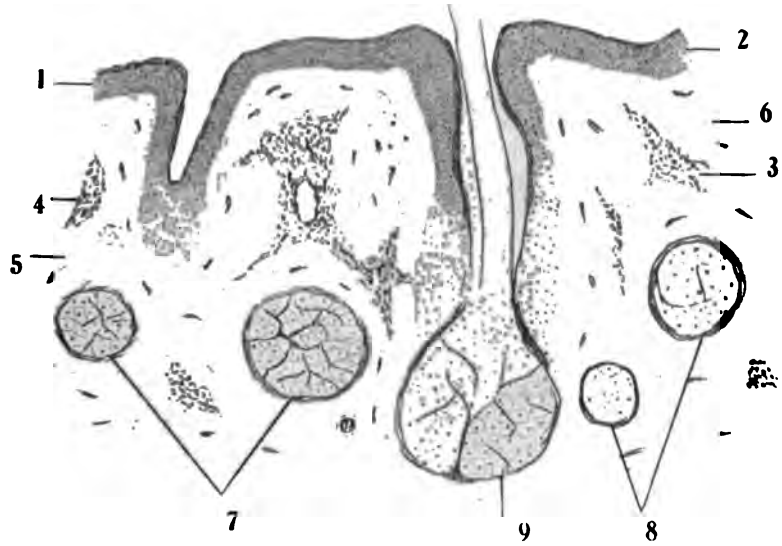


Fig. 200.—Microscopic section of a conjunctival dermoid tumor. (AUTHOR.)

(Original drawing by DR. CARL FISCH.)

1, 2, Epithelium of the conjunctiva. *3, 4*, Round-cell infiltration. *5, 6*, Connective tissue. *7, 8*, Sebaceous glands. *9*, Hair-follicle with sebaceous gland.

from the caruncle. They are composed of connective tissue and blood-vessels, and bleed easily. They are not smooth like the polypi, but present a papillary, nodulated, or cauliflower-like appearance. They have a broad base, and are likely to recur. In rare instances they undergo carcinomatous degeneration. *Granulomata* resemble polypi, but differ in this respect: they are not covered by conjunctiva, but are naked granulation masses. They arise from surfaces made raw either by ulceration or by operation. They are often found after an operation for strabismus, enucleation, or chalazion. They cause bloody tears and many of the miraculous instances of bloody lacrimation can be thus explained. They are soft, irregular on the surface, but often become smooth from

friction. They may expand so as to cover one-half the inner surface of the upper lid. They should be removed by the scissors. The base should be cauterized. If this be thoroughly done, they do not return.

Vascular Tumors.—Primary angiomata of the conjunctiva are very rare. They are congenital and increase in size after birth. They usually spring from the caruncle. Reik met with a cavernous angioma growing from the bulbar conjunctiva. The large angiomas found about the eye generally spring from the eyelid or from the depths of the orbit. They form dark purple-red tumors. Microscopically they are composed of loose connective tissue with many small areolar spaces and numerous blood-vessels. The large blood-spaces are traversed by connective tissue and are lined with endothelium. Angiomas should be excised. The author has seen one case of angioma of the retina involving also the tissues of the orbit and side of the face. Enlarged tortuous veins in the conjunctiva are often present in glaucoma and in the eyes of alcoholics. True varix is sometimes seen as a blue mass of veins, pyramidal in shape, the apex pointing toward the cornea. Phleboliths have been found in these

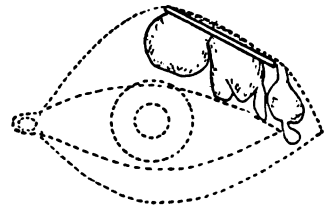


Fig. 201.—Polypi of the conjunctiva. (De Beck.)

The outlines of the cornea and the lids are shown by dotted lines. The upper lid is everted, and a foreign body, a piece of straw, is seen in the upper fornix.

masses. Pressure will empty these tumors, and ligation is the proper treatment.

Burnett has recorded an interesting case of hematoma of the conjunctiva. The tumor was the size of a pigeon's egg.

Osteoma of the conjunctiva is an unusual disease of which examples have been recorded by Critchett, Snell, Heustis, Oliver, and others. In the case treated by Heustis the piece of bone measured 7 by 4 millimetres. It was situated near the outer canthus. His patient was twenty-three years of age. Osteomata and fibro-osteomata of the conjunctiva are supposed to be congenital growths. They have been found in very young children. They should be excised.

Cysts of the conjunctiva are not rare. They appear usually in the lower lid as small vesicles of a straw color. They can be incised and curetted. The cysts found in the ocular conjunctiva arise generally from dilated lymph-vessels.

Subconjunctival Cysts.—A rare form of cyst is that which is due to enlargement of the acinotubular glands of Krause, which are most numerous in the fornices and in the caruncle. Gelatinous cysts

ituated beneath the ocular conjunctiva have been described by Fehr. he subconjunctival cyst shown in Fig. 202 was immobile and tense. It veloped beneath an overlying pterygium. When incised, a thin, straw- lored fluid escaped. The growth, which was of eight years' duration, ed the space between the cornea and the inner canthus. Microscopic mination showed a tissue richly supplied with spaces, which were ed with layers of epithelial cells resting upon a dense, thin, fibrous ucture. The general appearance was that of dermal structure, al- ugh sebaceous glands and hair-follicles were absent.

Cysticercus.—Large cysts beneath the mucous membrane arise m the cysticercus cellulosæ. They are generally found in children young subjects. Early in its history the cyst is transparent and e head of the animal may be seen. The overlying conjunctiva becomes flamed and opaque, thus making the diagnosis difficult. The most ommon site for such a cyst is beneath the ocular conjunctiva. Rarely

Fig. 202.—Subconjunctival cyst. (Author.)

is it found in the fornix or palpebral conjunctiva. Juda has recorded an interesting case of cyst of this character in a boy six years old. The cyst occupied the space between the cornea and caruncle. Cysticercus is rare in all countries except Iceland, Finland, northern Germany, and Manitoba. In northern Germany among 80,000 patients von Graefe saw 8 cases in the deeper parts of the eye, 3 in the anterior chamber, 5 beneath the conjunctiva, and 1 in the orbit. Few cases of cysticercus of any part of the eye have been seen in the United States. The treatment consists in incision of the conjunctiva and removal of the sac.

Lymphectasiæ.—The lymphectasiæ result from dilation of the lymph-channels of the conjunctiva. The lymphatics appear as small transparent beads, generally placed in the palpebral fissure, midway between the cornea and canthus. Leber has reported one case in which the lymph was periodically mixed with the coloring matter of the blood (lymphectasia menorrhagica). The lymphectasiæ cause little or no trouble. If treatment is required, they may be opened or excised.

MALIGNANT TUMORS OF THE CONJUNCTIVA.

Sarcoma.—A sarcoma of this region is either a leucosarcoma, with or without hematogenous pigmentation, or a melanosarcoma. The former

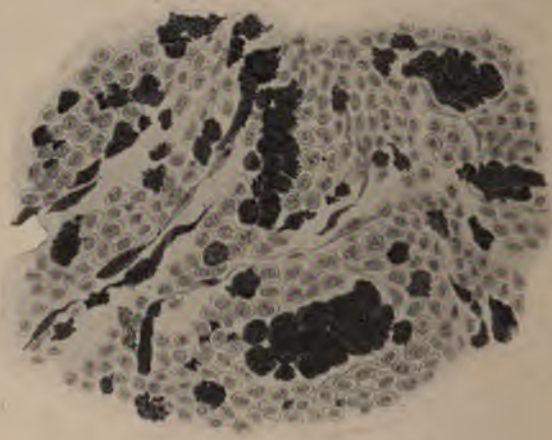


Fig. 203.—Microscopic section of an epibulbar leucosarcoma with hematogenous pigmentation. (KERSCHBAUMER.)

develops from the conjunctival or episcleral vessels, the latter from a nævus pigmentosus. The first sign of the tumor is a growth of the adventitial cells of the conjunctival vessels. When formed, the tumor-mass is

Fig. 204.—Antero-posterior section of primary sarcoma of the conjunctiva. (AUTHOR.)

The neoplasm extends over the cornea; the anterior chamber is obliterated. The detachment of the retina is accidental.

rich in vessels. The stroma shows spindle-shaped, round, or polygonal cells lying in close proximity. Cell-nests of varying size may be present, thus greatly resembling carcinoma. Melanosarcomas of the corneoscleral region

spring from nevi and often show a structure resembling that of nævus pigmentosus of the conjunctiva and skin. These so-called "alveolar forms" led Panas to class all melanotic tumors of the corneoscleral region as carcinomas—a view which has been shown by Kerschbaumer to be incorrect.

Early in its history a sarcoma causes no symptoms. After the growth has become larger there is pain and redness. The mass becomes anchored to the cornea and spreads over it. It may involve the whole conjunctiva

Fig. 205.—Growth of conjunctival epithelioma into the cornea. (De Beck.)

The epithelial growth is seen extending to either side of the limbus. The flat tumor, resembling a condyloma, is attached to the limbus by a central core of connective tissue.

Fig. 206.—Microscopic section of an epithelioma of the conjunctiva advancing into the cornea. (De Beck.)

At the right is the normal corneal epithelium; at the left this passes into the irregular cells of the new growth.

without penetrating the globe; or it may gain access to the interior of the eye by burrowing between the scleral lamellæ, or between the corneal layers, or by following the lymph-spaces of the anterior ciliary vessels. When the intra-ocular tissues are involved by an epibulbar sarcoma, increased tension is rarely observed. The melanotic form grows rapidly, often forming metastases. These black tumors are easily recognized. Epibulbar sarcomas occur chiefly in persons above fifty years of age. Three of the reported cases occurred in children. The author's case (Fig. 204) was that of a middle-aged negro.

Epibulbar leucosarcomas are often confounded with epitheliomas. The latter are flatter, are more cracked, and show a smaller lobulated structure; the sarcomas are elevated, and show a smooth surface or large lobules. The epitheliomas are solid, while the sarcomas are elastic. The former are likely to grow into the lid, while the latter are more apt to spread into the globe. However, it will often occur that the exact nature of a malignant tumor of the conjunctiva can be determined only by microscopic study. This is particularly true if the tumor is removed while yet small. Fortunately sarcoma of this region is rarely seen. Of 67 cases of sarcoma of the eye tabulated by Kerschbaumer, 9 were epibulbar; 5 of these were leucosarcomas, 2 were leucosarcomas with hematogenous pigmentation, and 2 were melanosarcomas. A peculiar form of sarcoma known as *cylindroma* has been observed in the conjunctiva.

It is possible to mistake an epibulbar sarcoma for tubercular granulation tumors, which pierce the corneoscleral region, or for an intra-ocular sarcoma piercing the sclera in front. The history of the case should suffice to prevent such a mistake. Danvers states that in some cases the differential diagnosis between the ocular form of spring catarrh and sarcoma of the conjunctiva can be made only by microscopic examination.

TREATMENT.—Any suspicious tumor growing from the conjunctiva should be subjected to an early and thorough removal. A microscopic diagnosis can then be made and the case can be carefully watched. If a sarcoma has involved the tissues of the globe, an enucleation will be necessary. The prognosis in these cases is favorable only if the operation be undertaken early. If the tumor involves the capsule as well as the globe, an exenteration of the orbit must be made.

Epithelioma.—This disease, attacking by preference those parts of the body where one epithelium joins another, as the lips, nose, etc., begins in the conjunctiva near the corneoscleral junction (Fig. 8, Plate VII). Here there is not so much difference in the epithelial covering as in the character of the underlying tissues. An additional factor in the development of this type of tumor in this region is the peculiar arrangement of epithelium at this place, for in normal sections the epithelium is often found growing into the corneal tissue in the form of conic processes.

The tumor begins as a small, rounded, or nodular, hard mass, slowly increasing in size and not painful. Eventually it spreads to all the ocular structures. Microscopically it is found that round-cell infiltration precedes the advancing growth of epithelial cylinders. The cylinders grow along the corneal canals. The corneal lamellæ become broken up and destroyed, Bowman's layer disappears, and the membrane of Descemet resists the longest. When it ruptures, the iris becomes fastened in the wound and the new growth spreads to the deeper structures. On the other hand, the epithelioma may spread along the lymph-sheaths of the anterior ciliary arteries. Often these tumors are lobulated and overlap the cornea, from which they can be lifted with a probe. The cornea may appear not to be

infiltrated, but only flattened by the mass, but microscopic section will show it to be involved. The surgeon, under these circumstances, shaves the growth off level with the cornea, touches the base with the electrocautery, and expects a cure. Almost invariably, however, there is a recurrence, but not necessarily at the old site. The microscopic appearances are represented in Fig. 206, where the normal corneal structure is shown on the right and the advancing apex of the tumor is seen on the left. These tumors are rarely pigmented. They are found in middle-aged and elderly persons. Although springing usually from the limbus, epitheliomas sometimes grow from other parts of the conjunctiva.

DIAGNOSIS may be difficult or impossible until after microscopic examination.

PROGNOSIS.—Epithelioma of the conjunctiva is always a serious disease. If removed early and thoroughly it may not return.

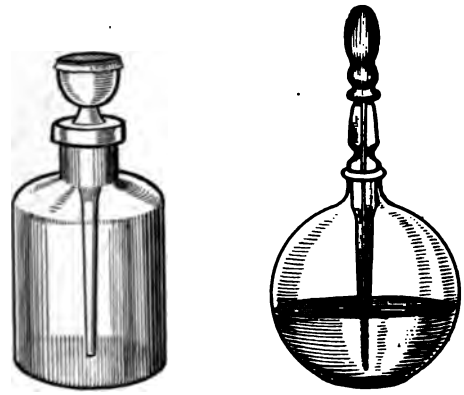


Fig. 207.—Eye-drop bottle (CHALK) and aseptic flask (STROSCHEIN).

TREATMENT.—When small, the cornea not being involved, the growth is to be excised. If large, or with considerable involvement of the cornea, the eye must be enucleated. Exceptionally cases occur in which removal of a tumor which involves the superficial layers of the cornea will not be followed by a recurrence. If the growth is large and has extended into the eyeball, it will be advisable to remove all of the orbital contents.

INFLAMMATIONS OF THE CONJUNCTIVA.

Inflammations of the conjunctiva are characterized by hyperemia; abnormal amount of secretion, which may vary from lacrimation to the production of mucus or pus; pain, which may be slight or severe; and dread of light (photophobia). The most noticeable of these changes, in case of a slight inflammation, is the increase in vascularity. It is necessary to distinguish the hyperemia of conjunctivitis from that which occurs in keratitis and deep-seated ocular diseases, such as iritis, chorioiditis, and iridochorioiditis. The former is known as *conjunctival,* and the latter

as *ciliary injection*. Although in severe forms of inflammation of the anterior ocular segment the two types of congestion are associated, as would be expected from the numerous anastomoses between the posterior conjunctival and the anterior ciliary vessels, yet in many cases they can be readily distinguished. The points of differentiation between these forms of hyperemia are as follows:—

Conjunctival Injection (Fig. 208).	Ciliary Injection (Fig. 209).
1. Comes from the posterior conjunctival vessels.	1. Comes from the anterior ciliary vessels.
2. Is found in conjunctival diseases.	2. Is found in diseases of the iris, ciliary body, and cornea.

Fig. 208.—Congestion of the posterior conjunctival vessels. (Author.)
(Original drawing by Dr. R. W. Mills.)

3. Is movable with the conjunctiva when pressure is made with the eyelid intervening.	3. Is immovable when the conjunctiva is moved.
4. The greatest redness is posteriorly, in the fornices.	4. Greatest redness is circumcorneal.
5. Redness lessens toward the cornea.	5. Redness lessens toward the fornix.
6. Color is a brick-red.	6. Is of a pink or lilac color.
7. Is composed of coarse, tortuous, superficial vessels whose meshes can be discerned.	7. Is composed of a series of fine, straight vessels radiating from the periphery of the cornea. The individual vessels are not easily recognized.

Hyperemia of the Conjunctiva (Dry Catarrh).—This may be active (acute) or passive (chronic).

Acute Hyperemia is the first change noticed in catarrhal or in purulent conjunctivitis. In this place, however, reference is had to idiopathic hyperemia and to that form which may be due to the temporary lodgment of foreign bodies in the conjunctival *cul-de-sac*. The causes are numerous, and include the lodgment of cinders and particles of dust; the exposure to irritants, such as smoke, chemicals, strong winds, and bright light; the abuse of alcoholic beverages; and the strain of an uncorrected or improperly corrected error of the muscle-balance or of the refraction. Beginning presbyopia is a frequent cause. Hyperemia is often found in the early stage of cataract and in small corneal opacities. In some cases

Fig. 209.—Ciliary injection. (Author.)
(Original drawing by Dr. R. W. Mills.)

of the gouty diathesis acute hyperemia, which may be recurrent, is found. Excessive and prolonged use of the eyes at near points, the presence of catarrhal or atrophic inflammation of the nasal mucous membrane, and disease of the accessory sinuses will cause vascular engorgement.

Symptoms. — These include a sensation of irritation or discomfort about the eyes. They feel as if "sand" were in them. They are heavy and their use soon causes them to tire. In case of the lodgment of a foreign body the eye becomes rapidly red and there is profuse lacrimation. Photophobia is a common symptom. Inspection shows congestion of the posterior conjunctival vessels.

Treatment.—From what has been said it is evident that search should be made for foreign bodies, nasal disease, and errors of refraction. The

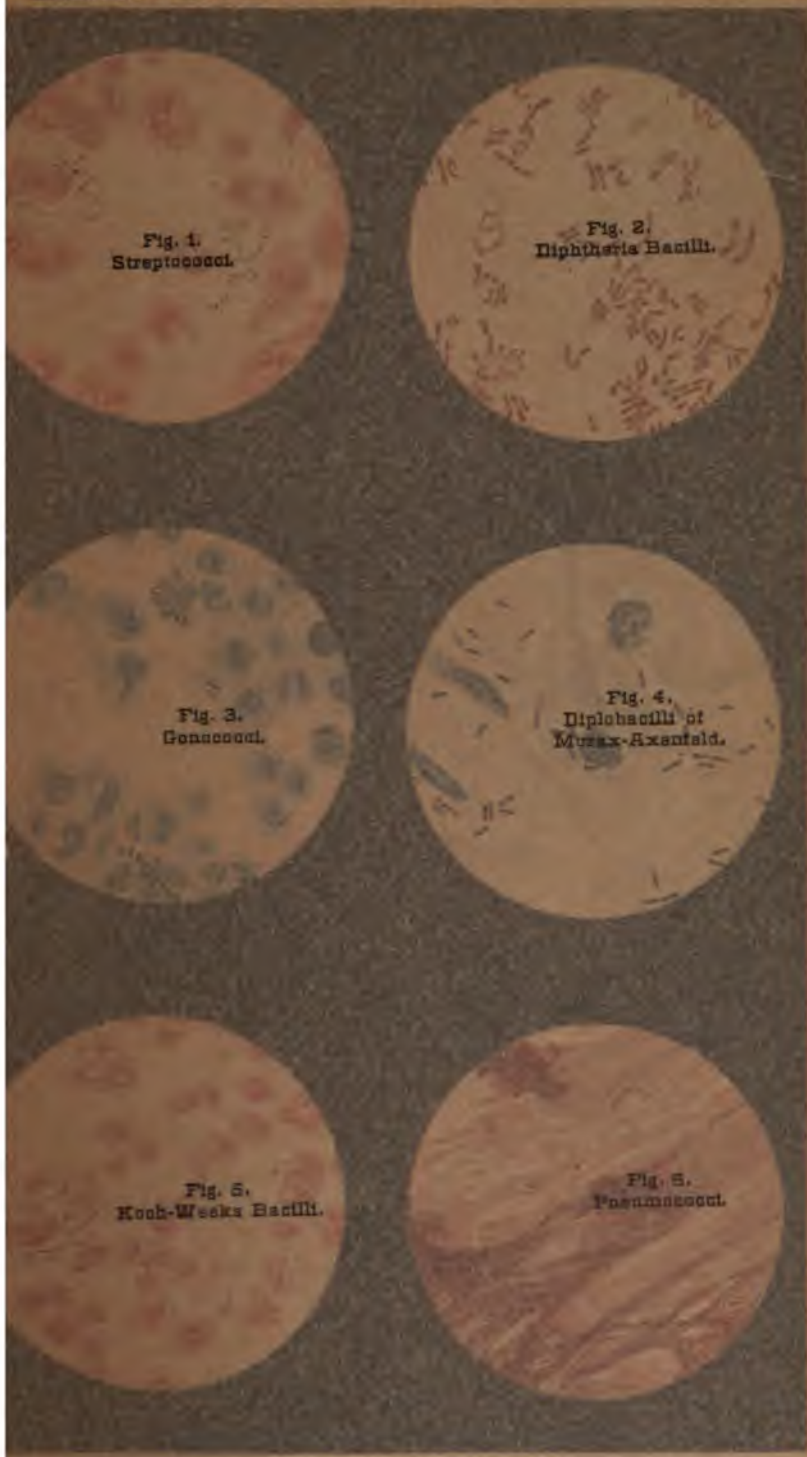
condition of the general health should be investigated. The correction of bad habits and removal from unhygienic surroundings will often be followed by immediate improvement. The local use of very hot water applied to the eyes for a few seconds thrice daily is beneficial. At night the patient can rub a small piece of the ointment of ammoniated mercury (1 to 20) upon the closed lids. The use of a mild collyrium—boric acid, bicarbonate of soda, or camphor-water—is often advisable. A favorite mixture is: zinc sulphate, gr. $^1/_4$ to $^1/_2$; cocain, gr. j; water, ℥j, of which 2 drops are to be used in the eye twice a day. The use of strong astringents is inadvisable. In applying a collyrium the surgeon everts the upper lid and drops the medicine upon it. If it is to be used at home, the patient should not attempt the eversion, but should pull the lower lid downward, at the same time looking down, while another person drops the medicine into the cup thus made. The lids can then be gently closed and kept closed for two or three minutes. The eye-dropper should always be held "right side up." Inattention to this point will result in the medicine running into the rubber bulb, and thus foreign bodies may be dropped into the conjunctiva along with the medicine. In the application of collyria the surgeon should use one of the numerous antiseptic droppers and bottles, of which those of Galezowski, Chalk, and Stroschein are among the best.

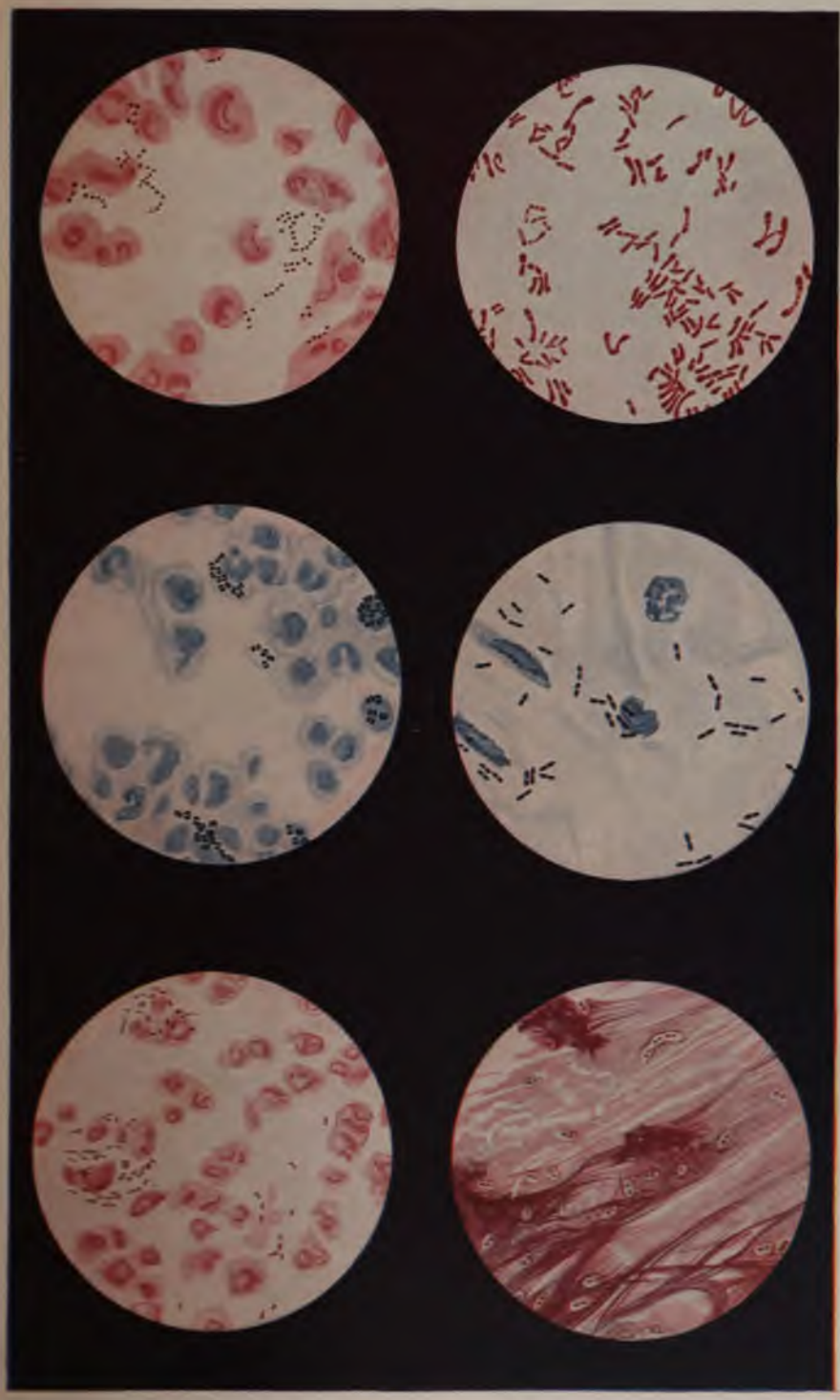
Passive Hyperemia.—In this form of congestion the venous circulation is interfered with, the veins being larger and more tortuous than under normal conditions. This often leads to hypertrophy of the papillæ of the conjunctiva. Passive hyperemia is found after paralysis of the cervical portion of the sympathetic nerve. It is common in gouty persons and in those who are ametropic. The proper treatment consists in the removal of the cause and the use of mild astringent collyria.

Conjunctivitis exists where the presence of an abnormal secretion is added to hyperemia. Almost all forms of conjunctivitis are contagious. Although they produce certain pathologic changes in the affected membrane, it must be remarked that there is often no definite and constant result following the transmission of secretion from a diseased to a normal eye. The same secretion, obtained from an eye the seat of conjunctivitis, may be inoculated into several healthy eyes and produce a different clinical picture in each. Hence it is supposed that the type of inflammation set up is influenced by other than the bacterial cause. The rôle of the toxins in inflammatory processes offers an inviting field for study. So long as the protective epithelium of the conjunctiva and cornea is intact, the patient is comparatively safe from microbic invasion (Randolph). Doubtless every form of conjunctivitis, except the chemical or mechanical, is due to bacteria, although it is yet too early to found a classification of the conjunctivitides solely upon bacteriologic causes. Hence, in the pages to follow the author will classify these affections from their clinical pictures, speaking of the concomitant bacilli and cocci as etiologic factors. According to the secretions present and the pathologic changes found, inflammations of the

PLATE IX.

Bacteria Found in Conjunctival Inflammations. (SCHANZ.)





conjunctiva are classified as catarrhal, purulent, diphtheritic, granular, and phlyctenular. As a rule, the prognosis of inflammation of the conjunctiva is favorable. Many cases, however, of the purulent and granular forms lead to corneal complications; and occasionally an eye is lost either from the neglect of the patient to consult a skilled surgeon at an early date, from injurious methods of treatment, or from the violence of the inflammation.

Simple Conjunctivitis (Catarrhal Conjunctivitis; Catarrhal Ophthalmia).—This is a mild form of conjunctivitis (Fig. 5, Plate VIII) which is accompanied by swelling of the membrane and lids and a muco-purulent secretion. The disease tends to spontaneous recovery. It begins with a burning or smarting sensation, which is more pronounced before the appearance of secretion. The conjunctiva, particularly in the region of the fornices, is hyperemic. In severe cases the conjunctiva is swollen; there is photophobia and increased lacrimation. Often the movements of the eye and accommodative effort will cause pain. One or both eyes may be involved. Vision is affected only when mucus adheres to the cornea.

Etiology.—The mechanical and traumatic varieties of simple conjunctivitis result from the presence in the *cul-de-sac* of small foreign bodies such as dust, pollen, insects, etc. The disease is associated often with the exanthemata, such as small-pox, measles, scarlatina, influenza, eczema, blepharitis marginalis, and facial erysipelas. It is a common accompaniment of acute coryza. Scrofulous subjects and persons having folliculosis are particularly susceptible to this disease. Streptococci, staphylococci, and pneumococci are present in severe cases. According to Gifford, the pneumococcus of Fraenkel is a common cause of simple conjunctivitis. On the other hand, Morax and Parinaud regard this bacterium as an infrequent cause of this form of conjunctival inflammation.

The pneumococcus of Fraenkel (known also as the diplococcus pneumoniæ of Weichselbaum, micrococcus lanceolatus of Talamon, and micrococcus Pasteuri of Sternberg) is in the form of oval cocci, often arranged in pairs or chains, with the free ends often pointed and encapsulated (Fig. 6, Plate IX).

Diagnosis and Prognosis.—From the foregoing description it will be noted that the diagnosis is generally an easy matter. If in doubt, a microscopic examination will be conclusive. The prognosis is favorable.

Treatment.—When the cause is removed these cases tend to spontaneous recovery. Cleanliness is an important factor in treatment. The eyes should be washed with hot water two or three times daily. A 1-per-cent. strength solution of boric acid, a $^1/_5$-of-1-per-cent. solution of chlorid of zinc, a 1 to 15,000 solution of bichlorid of mercury, or the zinc-cocain solution previously mentioned may be used twice daily with benefit. Protargol (5 to 10 per cent.), largin (5 to 10 per cent.), argentamin (2 to 5 per cent.), and argyrol (5 to 10 per cent.) are valuable remedies. Smoked glasses should be prescribed. *Poultices of any kind are absolutely contraindicated in all forms of conjunctivitis.* After recovery, the condition of

the refraction should be investigated and ametropia, if found, should be corrected.

Acute Contagious Conjunctivitis (Acute Catarrhal Conjunctivitis; "Pink Eye"; Epidemic Ophthalmia; Acute Muco-purulent Conjunctivitis).—This is an acute inflammation of the conjunctiva, having a definite period of incubation. It is accompanied by a muco-purulent discharge, and both eyes are usually affected. The disease is more common in the spring than in the fall months and often becomes epidemic. It is not limited to any country or climate. It is found at all ages except possibly during the first ten days of life.

ETIOLOGY.—Since Koch studied this disease in Egypt in 1883, finding a bacillus resembling that of mouse-septicemia; and Weeks in 1886 was able to make pure cultures of this bacillus and produce the disease in healthy conjunctivæ; and Morax, Hansell, and others confirmed these observations, great stress has been laid upon the Koch-Weeks bacillus as the cause. This bacillus (Fig. 5, Plate IX) stains readily with anilin dyes and often is associated with the xerosis bacillus. In the last few years, however, it has been shown that the pneumococcus is more often the cause of acute catarrhal conjunctivitis than is the bacillus just described. Gasparini in 1893 was the first investigator to note that the muco-purulent type of conjunctivitis could be caused by the pneumococcus. The investigations of Gifford, who found the pneumococcus in 36 of 40 cases, and of Junius, who met with it in 49 of 60 cases of acute catarrhal conjunctivitis, have gone far toward the settlement of the etiology of this disease. Gifford carried the disease to healthy eyes by means of the discharge, and always found the pneumococcus in the discharge from the inoculated eye. In 31 of the 60 examinations made by Junius the pneumococcus was found in a pure state; in 18 it was associated with the staphylococcus or with the xerosis bacillus. Recent investigations by Veasey show that for Philadelphia and vicinity the most frequent cause of acute catarrhal conjunctivitis is the pneumococcus of Fraenkel. Of 64 cases examined by this observer, the pneumococcus was present in 52. In 10 of these the pneumococcus was mixed with other bacteria. Those cases in which the micrococcus pyogenes albus or aureus was present were mild in character. In only three of Veasey's cases was the Koch-Weeks bacillus present. Occasionally the disease is produced by the Koch-Weeks bacillus. The clinical signs of both infections are so similar that in severe cases only a bacteriologic examination can distinguish between them.

Atmospheric influences are supposed etiologic factors, the disease being more common at those seasons of the year when coryza, influenza, and exanthemata prevail. The infection is often carried by washing utensils, such as towels and handkerchiefs, particularly in barracks, work-houses, orphan asylums, and hospitals.

PATHOLOGY.—There is great enlargement of the conjunctival vessels, apparent increase in the number of small vessels, thickening of the con-

junctiva, particularly in the fornices, from vascular engorgement and serous effusion, and slight infiltration of leucocytes at the base of the epithelial layer of the conjunctiva and between the cells. Pneumococci or bacilli are present in the discharge.

SYMPTOMS.—About thirty-six hours after infection the eyelids smart, and later the lashes are glued together with the drying of the discharge. The conjunctiva is hyperemic and the eye looks red and swollen. The discharge increases. At first it is chiefly mucus (normal secretion and shed epithelium), of which small, stringy masses float in the lacrimal secretion, and is of a gray or whitish color. When the proportion of pus increases, the discharge is yellowish, and may be so profuse as to simulate gonorrheal infection. In a few cases a pseudomembrane appears, and, in the absence of a bacteriologic examination, such cases may be mistaken for diphtheritic conjunctivitis. The eye is of a bright-red color; hence the name "pink eye." The patient often complains of the sensation of a foreign body, and in delicate nervous subjects pain may be a prominent symptom. Vision is blurred from the smearing of the discharge over the cornea. Use of the eyes causes pain. The disease usually reaches its height on the third or fourth day. The bulbar conjunctiva may be slightly or greatly involved, according to the severity of the process. When it is much swollen, with a large serous exudation lifting it up around the cornea, the condition is named *chemosis*. This may be so great as to cause the swollen tissue to protrude between the eyelids. In some severe cases small hemorrhages appear in the conjunctiva. The acute stage of catarrhal conjunctivitis lasts from five to ten days, the disease tending, in the majority of cases, to spontaneous recovery. If neglected, it may remain a long time in the subacute or chronic form. Corneal complications are rare. The development of corneal disease, characterized by an increase in pain and photophobia, begins at or near the limbus in the form of small, opaque, gray spots often arranged concentric with the corneal periphery. In a few days these points unite to form a concentric infiltration. Later the epithelium breaks down, and thus an ulcer is produced, which is known as a catarrhal ulcer. Generally it heals rapidly, leaving a minute scar, which from its location cannot interfere with vision. In rare instances such an ulcer leads to general corneal infection and perforation. Such a disastrous result is exceedingly rare in cases properly treated, but may follow improper treatment: *e.g.*, the use of poultices. In almost all cases both eyes are affected in acute conjunctivitis. In one the inflammation may be at its height, while in the other it is beginning.

DIAGNOSIS.—The bacteriologic diagnosis depends on the finding of the pneumococcus or the Koch-Weeks bacillus. The student or practitioner who is familiar with laboratory methods will have no difficulty in this respect. For the majority of practitioners, however, the clinical diagnosis will suffice. The presence of a discharge, the location of the greatest redness in the region of the fornices, and the character of the secretion, in the vast

majority of cases, will enable the practitioner to make the diagnosis without difficulty. It is only in the rare cases where the presence of a pseudo-membrane simulates diphtheritic or where a profuse purulent discharge resembles gonorrheal ophthalmia that doubt may arise. In such instances the history of the case and attention to the symptoms of these affections as described elsewhere in this chapter, will serve to clear the diagnosis. If any doubt remains, a bacteriologic examination should be made. It must be remembered that iritis may exist and be overlooked during an attack of acute catarrhal conjunctivitis (see "Iritis"). A more common mistake is to call iritis catarrhal conjunctivitis.

CLINICAL VARIETIES.—Attention must be called to certain forms of catarrhal inflammation which are of sufficient importance to deserve mention. Thus, *traumatic conjunctivitis* results from blows, the lodgment of foreign bodies, the action of chemicals, the irritating effect of dust (found among millers, masons, etc.), the effect of exposure to bright light (electric welding, snow-blindness), repeated exposures to the x-rays, and to inoculation of animal matter found around slaughter-houses. In such cases photophobia, lacrimation, pain, and swelling are likely to be symptoms of unusual prominence, and edema of the lids and superficial corneal ulceration are common. A severe form of the disease is observed in gas-fitters.

Exanthematous conjunctivitis is found in connection with eruptive diseases, particularly rubeola. *Follicular conjunctivitis,* which is classed by some authors with catarrhal and by others with trachomatous inflammations, will be considered separately. In the form of conjunctivitis known as *vesicular catarrh* the tarsal conjunctiva is covered with numerous small elevations, which Arlt compared to the appearance of fine sand scattered over a moist glass plate. A rare form is the *pustular conjunctivitis,* in which pustules appear on the bulbar conjunctiva and leave small grayish or yellowish ulcers. This disease resembles phlyctenular conjunctivitis. In the latter affection the conjunctiva of the fornices and lids is only slightly or not at all affected, while in pustular conjunctivitis they show signs of catarrhal inflammation. *Pink eye* is a form of epidemic catarrhal conjunctivitis affecting the lower animals as well as man, and is transmitted by the atmosphere. A mild form of catarrhal inflammation, bilateral, and found in gonorrheal or gleety subjects who have joint complications, has been named *metastatic gonorrheal conjunctivitis.*

PROGNOSIS.—Acute catarrhal conjunctivitis tends to recovery. It is only in cases of unusual severity, and in those in which poultices or other improper methods of treatment are used, that serious complications ensue. Ordinarily, under the use of cleanliness and astringents, the disease yields readily, and the patient can be discharged in from ten to fourteen days.

TREATMENT.—Removal to proper surroundings where the air is pure and free from dust is advisable. Cleanliness is of importance. The eyes should be bathed often in hot water. The chief remedy for catarrhal conjunctivitis is a solution of nitrate of silver applied to the everted lids once

a day. The strength of the solution should vary according to the intensity of the inflammatory process. Ordinarily a solution of the strength of gr. ij to ʒj will be sufficient, but for severe cases gr. v will be better. A stronger solution than this is never needed in the treatment of this disease. In using a solution of the strength mentioned it will be unnecessary to neutralize the excess of silver with a solution of chlorid of sodium. Between the daily treatments by silver the following collyrium, which is highly recommended by J. H. Thompson, of Kansas City, can be used: Boric acid, ʒiss; calcined magnesia, ℈ss; water, ʒiij. Mix and filter. A few drops of this can be used in the eye every four hours. When chemosis and edema are marked symptoms the application of cold compresses for a few minutes will be useful. In such cases some ophthalmic surgeons favor the direct application of ice to the closed lids. From its blanching properties it might be supposed that a 2- to 5-per-cent. solution of the extract of suprarenal capsule would be useful when dropped into an inflamed conjunctiva, but its effect is only temporary, and the inflammation returns with force equal to or greater than before. Adrenalin chlorid (1 to 10,000) may be used with benefit. It is a powerful astringent.

Protargol has lately come into extensive use in conjunctival inflammations. It possesses no advantages over silver except that its application is attended by less pain. It is used in 5-, 10-, and 20-per-cent. strength solutions. Largin in 10-per-cent. solution is highly recommended by Sydney Stephenson as a substitute for nitrate of silver. Argyrol (5 to 20 per cent.) is a valuable remedy. Sulphate of zinc (gr. j to ʒj) is a valuable astringent, and can be used three times a day. Acetate of lead in the same strength is an excellent remedy, but in many cases it cannot be used, for the reason that, wherever the corneal epithelium has been removed this agent will form an indelible white deposit of lead carbonate, and thus will impair vision. An excellent prescription for acute catarrhal conjunctivitis is this: sulphate of hydrastin, gr. v; boric acid, gr. ij; biborate of soda, gr. iv; deodorized tincture of opium, ʒss; water, to make ʒj. Of this a few drops are used three times a day. Cuprol in 10-per-cent. strength solution, applied once daily, has been recommended. To preserve the efficiency of the drug a small amount of chloretone (0.5 per cent.) should be added. The author's experience with cuprol has been unsatisfactory. In prescribing collyria distilled water is often ordered when filtered water is better, since distilled water acts injuriously on epithelial cells (Thompson). Morphin is a remedy which is often prescribed for catarrhal conjunctivitis under the mistaken idea that it is soothing to an inflamed surface. It should not be used. If the pain is severe it is better to use a few drops of cocain, or, if pain is due to corneal infiltration, atropin. Cocain, however, should be used only at long intervals, since it first contracts and then paralyzes the blood-vessels. A better agent is holocain, $^1/_2$-per-cent. strength solution, which does not injure the vessels and has some antiseptic value. As a rule, the momentary application of very hot water will relieve the pain of con-

junctivitis. The presence of corneal ulcers in catarrhal conjunctivitis does not constitute a contra-indication to the use of silver, but the surgeon should take care that the medicine shall not come in contact with the cornea. If chemosis is great the swollen conjunctiva should be anesthetized and numerous punctures should be made in it with a cataract-knife. Bandaging of the eyes and the application of poultices are absolutely harmful in this disease. The patient should be given a pair of smoked glasses, and should get out of doors. The use of tobacco (smoking) and alcohol should be forbidden while he is under treatment.

Subacute Catarrhal Conjunctivitis (Subacute Conjunctivitis of Morax-Axenfeld) is found in all ages, but is particularly frequent in old age. The onset is sudden. There is a slight muco-purulent discharge, and a sensation of smarting without pain. The disease is usually bilateral, and does not lead to corneal complications. It can be distinguished from mild and prolonged cases of acute contagious conjunctivitis by the bacteriologic findings. In the former disease the pneumococcus of Fraenkel or the Koch-Weeks bacillus is present, while the latter is attributed to a particular microörganism which has been studied by Morax, Axenfeld, and Gifford. It is a diplobacillus, two or three micromillimetres long and one-half as thick, appearing as two short rods separated by a clear space, and can be distinguished from the pneumococcus by having no capsule and appearing more rod-like (Fig. 4, Plate IX). Gifford, however, believes it to be encapsulated. Morax found that a pure culture of it, carried through the fifth generation after incubation, causes typical inflammation in the human conjunctiva, but is not pathogenic for animals. It stains with dilute carbol-fuchsin and is decolorized by Gram's method. Eyes infected with this germ give considerable variety in the clinical pictures presented, although in most cases the symptoms are subacute or chronic. The daily use of a $^1/_5$-per-cent. solution of chlorid of zinc readily cures the disease.

Chronic Catarrhal Conjunctivitis.—This is a common disease which presents chiefly subjective symptoms. Objectively there is a moderate amount of hyperemia in the region of the fornices, or on the tarsus, or in both places. In many cases the conjunctiva is not swollen, while in those of long standing it looks velvety and is somewhat thickened. There is present a small amount of secretion, which is noticed chiefly by a matting together of the eyelashes in the morning, or by the presence of a dark crust at the inner angle of the eye. When this dried secretion is removed a thread of viscid mucus comes with it. The subjective symptoms include a feeling of heaviness or a sensation of sand in the eye, which symptom is more pronounced toward night; a burning or itching of the eyes, which tire quickly on accommodative effort; blurring of the sight, or the appearance of a rainbow around a light, caused by the spreading of mucus over the cornea; and often an annoying blinking and sensation of dryness. The disease is common among adults and elderly people and is likely to be of long duration. Frequently blepharitis marginalis is present. The skin

of the lids becomes eczematous, and ectropion is common. This increases the flow of tears, and the effort of the patient to wipe these away with his handkerchief adds to the local irritation. Corneal ulcers often occur in these cases.

ETIOLOGY.—The causes of chronic catarrhal conjunctivitis are numerous and include such dissimilar elements as failure to recover from an attack of acute inflammation, unhygienic surroundings, the injurious influence of certain vocations, abuse of alcohol and tobacco, exposure to wind and dust, eyestrain from an uncorrected error of refraction, the presence of blennorrhea of the lacrimal sac, and the existence of nasal disease.

TREATMENT.—If possible, the surgeon should first remove the cause. Under hygienic surroundings and mild treatment these cases show a rapid improvement. The eyes should be bathed frequently in hot water. The most popular remedies at present are argyrol, protargol, and nitrate of silver. The first and second are used in 5- or 10-per-cent. strength solutions, the third in the strength of $^1/_2$ to 2 grains to the ounce. Mild cases improve rapidly under the use of the zinc-cocain solution, or a 1-per-cent. strength solution of boric acid. The use of the ointment of ammoniated mercury, rubbed on the lid-margins at night, is beneficial. The collyrium should be applied to the everted conjunctiva by a dropper or by a mop. After the lids show signs of improvement the refraction should be examined. Often it will be found that hypermetropia or astigmatism is present in amount sufficient to explain the symptoms.

Follicular Conjunctivitis (Folliculosis; Follicular Trachoma, or Catarrh, or Ophthalmia; Simple Granular Conjunctivitis).— In children chronic catarrhal inflammation of the conjunctiva appears generally in the form of folliculosis (Fig. 4, Plate VIII). The disease has been so named because of the enlargement of the adenoid tissue, which takes the form of small, round, or oval translucent bodies, which are seldom larger than a rapeseed and resemble follicles. They lift up the conjunctiva, forming small elevations, and are more numerous in the lower than in the upper fornix. Often they are arranged in rows parallel with the folds of the membrane. Under a magnifying glass they look like little spots of jelly. They ultimately disappear under treatment, leaving the conjunctiva intact. In this respect they differ from trachoma. They are sometimes found in acute catarrhal conjunctivitis. This form of conjunctivitis is to be regarded as an expression of a tendency toward adenoid enlargement. The condition is extremely common. Thus, of 14,797 children, whose ages ranged from 2 to 19 years, Sydney Stephenson found only 5.55 per cent. with normal conjunctivæ; 93.99 per cent. presented folliculosis, and 0.46 per cent. had trachoma.

The question of the relationship between folliculosis and trachoma has caused much controversy. It is now generally accepted by ophthalmic surgeons that they are clinically distinct diseases, although histologically there is no line of demarcation between fresh follicles and fresh trachoma bodies.

In the absence of discharge from the eye folliculosis is not contagious. It tends to a spontaneous recovery without scarring of the conjunctiva. It is not associated with corneal complications, or with structural changes in the tarsal plate, and it never causes trichiasis, entropion, or ectropion. Trachoma, on the other hand, is always conditionally contagious, and is often associated with pannus, corneal ulceration, trichiasis, entropion, or ectropion. Folliculosis is rare after the twentieth year, while trachoma may be seen at any age.

The appearance of the conjunctiva in each affection may be described as follows (Stephenson) :—

False, or Follicular, Granulation.	True, or "Sago-grain," Granulation.
Oval or roundish, transparent bodies, the diameter of which seldom or never exceeds 1 or 1.5 millimetres. They often show a faint-yellowish hue, and are usually arranged in rows. Their tendency is to remain discrete: that is, separate from one another. They are always larger and better marked in the lower fornix.	Round, opaque, ill-defined bodies, of grayish-white color, and extreme friability. They are firmly and deeply imbedded in the conjunctiva, their diameter frequently reaching 2 millimetres or more. Their tendency is to become confluent, thus forming areas of trachomatous material. They are always larger and more numerous in the upper fornix.

Etiology and Pathology.—Follicular conjunctivitis is often said to be caused by unhygienic surroundings, but Stephenson has shown that practically no difference exists in the percentage of cases of folliculosis among the inmates of schools of good, medium, and poor social *status*. Folliculosis is often found among the children of farmers, among whom there is no lack of fresh air and sunlight. Among children, sex and age are without etiologic meaning. The disease, however, is often found in individuals who have enlarged cervical, femoral, or axillary glands, hypertrophy of the tonsils, adenoid growths in the pharynx, granular pharyngitis, and swollen gums. The conclusion seems evident that folliculosis is to be regarded as an expression of adenoid activity incident to childhood and youth. Some authors hold that the disease is contagious; others, that it is caused by vitiated air. The prolonged use of eserin will cause it (Juler). It is also seen after the use of atropin, particularly after the use of impure solutions. Some observers hold that dry atropin will cause it. It is probably due to a germ, but this opinion is only a supposition.

Pathology.—Microscopic section of the enlarged follicles shows them to be composed of a mass of lymphoid cells, held by a network of connective tissue, forming an incomplete capsule.

Prognosis.—The prognosis of folliculosis is favorable as regards vision, and so far as ultimate return to the normal condition of the conjunctiva is concerned; but the disease often is rebellious to treatment. In the course of years the follicles may disappear without treatment.

Treatment.—In considering the treatment of this disease it is advisable to distinguish between (1) cases without and (2) those with

catarrhal symptoms. In the first class the conjunctiva often is pale, but may show arborescent vessels. It presents the oval vesiculo-grains which have been described, and there is no discharge from the eye (folliculosis of Adamiuk). Aside from attention to the general health these patients need no treatment. If the follicles are unusually abundant, the daily use of a ½-per-cent. strength solution of silver nitrate, or a 10-per-cent. solution of argyrol, is advisable; or the surgeon can express the contents of the follicles with the roller forceps, a procedure which greatly shortens the period of treatment.

The second class presents a mucous or muco-purulent discharge, which is rarely abundant, but is in sufficient amount to cause the eyelashes to be glued together. The conjunctiva shows the vesiculo-grains. It is somewhat reddened and slightly thickened (follicular conjunctivitis). This form demands treatment. Here nitrate of silver or argyrol is indicated. Stephenson has obtained good results from the use of a carefully prepared ointment of the subacetate of lead (1 per cent.), of which a small piece is applied to the everted conjunctiva once a day, followed by massage. After two weeks' time the strength of the ointment is to be doubled. Under this treatment the redness disappears, the discharge ceases, and the follicles become reduced in size and finally disappear. The use of bluestone and strong solutions of silver is not advisable in this disease. Follicles which refuse to yield can be subjected to expression, or each follicle can be touched with the fine point of the galvanocautery. This method of treatment requires a tractable subject and great patience on the part of the surgeon. After the disappearance of the disease it will be well to search for errors of refraction.

Drug Conjunctivitis (Atropin Conjunctivitis) is the name which may properly be applied to those uncommon cases of conjunctival follicles which are caused by the application of certain medicines (atropin, cocain, homatropin, hyoscyamin, duboisin, eserin, and arecolin) to the conjunctiva. The toxic effect may follow a single application of the drug or may come only after prolonged use of it. In the former case the skin of the lid becomes dry, red, and swollen and may appear like erysipelas. Examination will show the presence of follicles, which are particularly numerous in the lower fornix. This type of the disease is more often found in adults than in children.

In the second form, after long use of a mydriatic or miotic, there is the sudden appearance of an acute catarrhal conjunctivitis with muco-purulent discharge. Inspection shows the presence of follicles.

Etiology.—Glorieux considers that the affection is a paralysis of the vasomotor nerves, with sequent dilation of conjunctival vessels. Idiosyncrasy must be a factor in some cases. A frequent cause is the presence of germs in the solution used or the existence of a chemical impurity. Some cases are caused by the transference of septic material by unclean eye-droppers.

TREATMENT.—The use of the drug should be discontinued and hot applications of the dilute subacetate of lead can be used. De Schweinitz uses a 1-per-cent. strength solution of creolin in atropin conjunctivitis. Tannin and glycerin solutions are valuable remedies. The skin of the lids should be smeared with the ointment of ammoniated mercury (1 to 20).

Vernal Conjunctivitis (Spring Conjunctivitis; Fruejahr's Catarrh; Phlyctenula Pallida; Circumcorneal Hypertrophy of the Conjunctiva).—This is a rare form of chronic catarrhal inflammation (Fig. 6, Plate VIII) which is characterized by hypertrophy of the conjunctival epithelium, the presence of an exudation into the corneoscleral margin, and the presence of peripheral opacities of the cornea. The disease was first described by Arlt, in 1846.

SYMPTOMS.—The patient complains of the usual symptoms of conjunctivitis. Burning or itching sensations and heaviness of the lids are frequent symptoms. Sometimes photophobia exists. Inspection shows the presence of a slight mucous discharge and the existence of lacrimation, hyperemia, and hypertrophy. The conjunctiva presents a bluish-white color resembling a film of milk spread over the membrane. It is thickened, the papillæ are prominent, and the hypertrophy causes large, flat-topped elevations of a reddish-gray color. They are found in the upper lid and around the cornea. In England and France the tarsal type is prevalent, while in Italy and other Mediterranean countries the bulbar form is more often observed. The limbus may show a jelly-like band around the cornea; or minute red dots may be present in the space corresponding to the palpebral fissure. In some cases only that part of the bulbar conjunctiva is involved which corresponds to the opening between the lids. In some patients the hypertrophies are few in number, resembling mushrooms, while in others they are abundant. The lesions may exist chiefly in the tarsal region or in the palpebral area. The patient has a dull, sleepy look, owing to the slight ptosis which is present. A clinical feature is the fact that the disease generally begins with the advent of warm weather in the springtime and continues during the summer and fall. It then disappears to return the following spring. Occasionally it persists during all the seasons.

ETIOLOGY.—The disease is almost limited to children between the ages of five and fifteen years, but when once set up the process may continue indefinitely. Both eyes are generally affected. Its etiology is unknown. Malaria and uterine disorders are supposed to be etiologic factors. The disease is of frequent occurrence in southern countries, but latitude alone is not a determining factor. Vernal conjunctivitis appears during the spring and summer months. It occurs in all grades of society and is not influenced by occupation or by exposure to the rays of the sun. The disease is sporadic and non-contagious.

DIAGNOSIS.—Spring conjunctivitis is to be differentiated from trachoma and from phlyctenular conjunctivitis. Spring conjunctivitis should not be confounded with the conjunctivitis attending hay fever. The

latter type of cases presents more of the anatomic and few of the symptomatic points found in vernal conjunctivitis. The pericorneal elevations of vernal conjunctivitis are permanent or of long duration, and they do not break down and leave ulcers. These characteristics will prevent the confounding of this disease with phlyctenular conjunctivitis. The differentiation from trachoma can be made by attention to the following table, which is by Danvers:—

Vernal Conjunctivitis.	Trachoma.
Occurs in all grades of society.	Is essentially the heritage of the poor.
Granulations are situated on surface of tarsus, and consist of fleshy, flat growths, with pedicles and well-marked grooves between them.	Granulations seem to be deep in substance of tarsus; oval in shape; of a grayish, transparent tint; and are seated more especially in the fornix, which they cause to atrophy.
Growths never ulcerate on bulb or tarsus.	Ulceration rare.
Pannus or corneal ulcer a rare exception.	Gives a special form of pannus beginning on upper part of cornea; corneal ulcer frequent.
Conjunctiva of tarsus has a bluish-white tinge.	Conjunctiva never bluish white, but a bright or dark red.
Disease heals without leaving any traces on cornea, tarsus, or fornix.	Cicatricial tissue on tarsus and fornix always left; after pannus there may be fixed opacity of the cornea.
In treatment, copper sulphate, nitrate of silver, and mercurial preparations always irritate the growths, and do no good whatever—in fact, are positively harmful.	All these substances are beneficial in the treatment of this disease.

Frequently the bulbar type of vernal conjunctivitis, with a single tumor-mass, can be differentiated from sarcoma and epithelioma only by a microscopic examination.

Pathology.—Microscopic sections of parts of the affected area show a proliferation of epithelial cells and the presence of lymphoid cells with bands of connective tissue in the large granular bodies found on the conjunctiva.

Prognosis.—The prognosis is favorable in the sense that the disease does not cause serious lesions of the cornea. It is a disease of long duration and may recur every summer for many years. The bulbar variety offers a more favorable prognosis than the tarsal type.

Treatment.—Many remedies have been proposed, but the most that can be done will be to relieve the distressing symptoms and stop the encroachment of the disease upon the cornea. A complete recovery often follows after several years. The eyes can be bathed in hot boric-acid solution, and ointment of yellow oxid of mercury or the ointment of ammoniated mercury can be rubbed into the conjunctiva once or twice a day. Bichlorid of mercury, zinc, silver, and other similar preparations can be tried in the

form of solutions. Van Milligen, who saw much of this disease in Constantinople, advises the use of a solution of acetic acid (from 1 to 20 grains to the ounce), but this remedy is not well borne. Magnani, of Turin, and Gallenga, of Parma, use ice-cold packs, which are applied to the eyes for ten minutes at a time five or six times a day. Danvers lays stress upon the examination of the nose and pharynx as preliminary to the ocular treatment. Darier states that in vernal conjunctivitis silver and copper are valuable only when there is an abundant, stringy, muco-purulent discharge, and that the pericorneal form of the disease is to be treated by massage with mercurial ointment made with lanolin. Natanson and Bock report cases in which powdered xeroform was used with excellent results. Randolph uses salicylic acid in solution (1 per cent.) and as an ointment. If there is much pericorneal injection atropin and smoked glasses are to

Fig. 210.—Section of growth in the bulbar type of vernal conjunctivitis. (Danvers.) × 100.

The epithelial covering is proliferated and sends long, thin processes into the connective stroma. In transverse section the processes resemble the nests of epithelioma.

be used. Attention to the general health is of importance, and often course of quinin, arsenic, or iron will be needed.

Surgical treatment of vernal conjunctivitis is admissible in the tarsal type. The lid should be everted, and the growths should be cut off with scissors. The hypertrophies around the cornea have been excised, burned by the electrocautery, or destroyed by electrolysis, but the results have not been satisfactory. If a single tumor of the bulbar conjunctiva is present it should be removed for diagnostic purposes. (Danvers.) Brossage is employed by some surgeons.

Purulent Conjunctivitis (Purulent, or Gonorrheal, Ophthalmia; Ophthalmia Neonatorum; Ophthalmoblennorrhea Neonatorum).—Ordinarily by these terms is meant an acute contagious conjunctivitis due to the gonococcus of Neisser, occurring either in infants within a few days following

birth or in adults. In the former the disease is known as the ophthalmia *of the* newborn; in the latter it is called gonorrheal ophthalmia. It has *been* shown recently by Groenouw that the bacillus coli communis, asso*ciated* with other microörganisms, was present in six of forty cases of oph*thalmia* neonatorum. Bietti has reported a case of blennorrhea in a new*born* child in which only the colon bacillus was found. The staphylococcus *and* streptococcus in exceptional cases are known to cause purulent oph*thalmia* indistinguishable by clinical signs from mild cases caused by the *gonococcus*. Rapidly extending necrosis of the cornea with abundant *purulent* secretion may be due to infection by the Klebs-Loeffler bacillus in *cases* of localized diphtheritic conjunctivitis. In view of these facts it *becomes* necessary to modify the definition of ophthalmia neonatorum. *The* medico-legal importance of these observations should not be overlooked *by the* surgeon, who, in the absence of a careful bacteriologic examination, *is not* justified in making positive statements as to the etiology of a case *of pu*rulent ophthalmia. Hence the subject of purulent ophthalmia will *be con*sidered under these heads: (1) gonorrheal ophthalmia of the adult; *(2)* gonorrheal ophthalmia of the newborn; (3) purulent ophthalmia not *due to* the gonococcus.

Gonorrheal Ophthalmia of the Adult.—This disease, which occurs more *frequ*ently in males than in females, is due to inoculation of the conjunctiva *with* secretion from a mucous membrane which is affected with inflamma*tion* due to the gonococcus of Neisser. The pus from a vaginal or urethral *gonor*rhea, or the discharge from a gleet, is conveyed to the eye by such *media* as the fingers, handkerchief, towel, or washbowl. It is improbable *that* the gonococcus is carried by the air. Gonorrheal ophthalmia of the *adult* is always a serious disease. It is characterized by great swelling of *the li*ds, a copious secretion of pus, a tendency to involvement of the cornea, *and* marked constitutional disturbance.

Symptoms.—The period of incubation ranges from a few hours to *three* days. This is followed by the period of infiltration, lasting two or *three* days, which begins with the signs of an acute catarrhal conjunctivitis. *Soon* the eyelids become hard, swollen, and brawny, the upper lid often be*coming* enormously enlarged and hanging over the cheek. The conjunctiva *of the* lids and fornix becomes much swelled, and often minute hemor*rhagic* spots are seen inside the lids. The conjunctiva is hard, rough, and *granular* from infiltration of seroplastic lymph, and presents a deep-red, *velvet*y appearance. The ocular conjunctiva is greatly swelled and forms a *ring* around the cornea (chemosis) and may protrude between the lids. *During* the acute stage the discharge is watery and sanious, and is streaked *with* flakes of mucus. There is great pain in and around the eye. The *pre-auricular* glands are tender and swollen and may suppurate. There is *a rise* of temperature, both local and general, and often the patient becomes *much* weakened by the inflammation. During this stage eversion of the *lids* is impossible without the outer canthus be cut.

In the stage of purulent discharge the lids become less hard and tender, the secretions are distinctly purulent, and the inflammatory symptoms are less marked. Enormous quantities of yellow or yellowish-green pus ooze out of the conjunctival sac and flow down upon the cheek. This stage continues for two or three weeks, and is succeeded by the stage of convalescence or papillary swelling. The conjunctiva may return gradually to the normal condition or may pass into a state of chronic blennorrhea, in which there is general redness and thickening of the membrane, with the presence of enlarged papillæ. The entire course of the disease lasts six or eight weeks.

This disease presents various clinical pictures according to its severity. As a rule, the mildest cases are those due to gleet; the severest result from infection with pus from a violent gonorrhea, the discharge from the vagina or urethra being thick and yellowish. In severe cases a croupous deposit may be seen on the conjunctiva; in mild cases its surface is covered with flakes of lymph, which are easily detached. In the severest cases a deep infiltration exists like that which is seen in diphtheritic conjunctivitis.

Etiology and Diagnosis.—The disease is due to the gonococcus of Neisser (Fig. 3, Plate IX), which occurs as a diplococcus and sometimes in a tetracoccus form. It is readily stained with aqueous solutions of aniline dyes, and is decolorized by Gram's method. Chartres states that the serious ophthalmias are those produced by streptococci, or by streptococci and gonococci, or by a combination of these with other bacteria.

The diagnosis of gonorrheal conjunctivitis is usually easily made by the history of the patient and by the symptoms described above; in case of doubt a bacteriologic examination should be made.

Complications.—These are numerous, and may be immediate or remote. That which is most dreaded is corneal involvement. This structure may present a dull hazy look a few days after the commencement of the inflammation. Small oval ulcers form around the limbus. These may be comparatively clean or may be surrounded by a zone of infiltration; or large central ulcers may be found. The corneal lesions may heal or may go on to perforation. They are due to two factors: pressure on the vessels and infection. Perforation of a central ulcer permits the escape of aqueous humor; the lens comes forward, closing in the opening, which is sealed by the deposition of lymph. Reaccumulation of aqueous is then followed by reposition of the lens, which carries with it a tag of lymph, thus forming an *anterior polar cataract.* Sometimes the iris becomes adherent to the posterior surface of the cornea, producing the condition known as *anterior synechia.* If the perforation occurs in an ulcer situated peripherally, the iris is sure to become adherent in the scar following the healing process, and thus is established an *adherent leucoma.* Perforation may be followed by infection of the ciliary body and chorioid, ending in destruction of the eye by *panophthalmitis,* or in the slow shrinking of the globe, which characterizes *phthisis bulbi.* The inflammatory process weakens the cornea, and often the intra-ocular pressure, acting on such a softened structure, leads

to *staphyloma* (see "Cornea"). In a few cases iritis occurs apart from perforation.

PROGNOSIS.—The prognosis of gonorrheal ophthalmia, which is always a serious disease, will depend on the violence of the inflammation, the intelligence of the surgeon, the faithfulness of the nurse, and the character of the treatment. An incompetent surgeon will lose nearly all the eyes he treats; a competent one will occasionally have a bad result. In young persons the prognosis is more favorable than in elderly individuals. The greater the chemosis, the greater the danger of corneal complications. Marginal ulcers are not so serious as central ones. Much can be foretold by the appearance of the cornea. If it be cloudy early in the disease, the outlook is bad; if milky, the cornea surely will slough. Sudden pain in the course of gonorrheal ophthalmia means a perforation of the cornea or the establishment of iritis. If the eyeball is not perforated and lost, vision may be so much reduced by scarring of the cornea as to be practically valueless.

PROPHYLAXIS.—Whenever called to treat a case of gonorrhea the surgeon should warn the patient of the danger of infecting his eyes. If one eye is already infected, the other should be immediately sealed up by means of Buller's shield, which is a watch-glass placed between two pieces of strong adhesive plaster or pieces of rubber. This is placed over the unaffected eye and is securely sealed above and on the nasal and temporal sides, leaving an aperture below for the air. Wherever possible, cases of gonorrheal ophthalmia should be isolated.

TREATMENT.—It may happen that the surgeon gets his eye infected when examining a patient, or is present when the accident happens to another. In such an event the conjunctiva is to be washed out with boric acid solution, and a strong solution of nitrate of silver (gr. x-xxx to ℥j) is to be applied to the membrane. This treatment may abort the disease. It is applicable, however, only to the period immediately following the infection.

The treatment of a case of gonorrheal ophthalmia will depend upon the stage of the disease. In the period of infiltration cold applications are useful. They should be applied day and night in this manner: Pieces of linen, or gauze, each two inches square and several layers in thickness, are to be placed on a cake of ice near the patient's bed. Every two minutes the pieces are changed, the one removed from the eye being destroyed. In mild cases it will suffice to use this treatment only during the day. Pads of absorbent cotton can be used in place of linen. The patient's bed should be narrow and capable of being approached from either side. The room should be well ventilated and scantily furnished. Vessels should be at hand for the collection and immediate destruction of all materials coming in contact with the discharge.

Aside from the use of cold and attention to the general condition of the patient, there is little to be done in the acute stage. The bowels

should be kept open. The pulse can be kept down by aconite or veratrum. When pain is severe one of the opium preparations should be used. Where the lids are greatly swollen a canthotomy will be in order. When this operation is necessary a general anesthetic should be given. With a pair of strong scissors the tissues at the outer canthus are to be cut down to the bone. The chemotic conjunctiva can then be felt. If hard, it is to be scarified in lines radiating from the cornea. This treatment relieves the blood-vessels and removes the pressure on the eye. The *cul-de-sac* can then be irrigated with warm boric acid (1 per cent.) or bichlorid solution (1 to 4000).

In the stage of purulent discharge the chief indications for treatment are: (1) cleanliness and (2) the use of germicides and reducing agents. The cold applications are now discontinued and the surgeon aims to keep the pus from accumulating by cleansing the conjunctiva every half-hour or hour during the time that the secretion is abundant. This will require both a day and a night nurse. The eye is to be cleansed by gently separating the lids, mopping up the pus with absorbent cotton, and washing the *cul-de-sac* with a boric-acid solution by means of a pipette. Many ingenious instruments have been devised for lifting the eyelid and at the same time throwing a stream of water on to the conjunctiva. All such contrivances are to be avoided as dangerous to the cornea in the hands of all except possibly their inventors. The kind of antiseptic solution used is of little moment as long as it is not too strong. The chief indication is to prevent the accumulation of pus. Boric acid (3 per cent.), bichlorid of mercury (1 to 15,000), or permanganate of potassium (1 to 5000) may be used with equal success. The solution chosen should be warm.

After the stage of infiltration is over and the lids can be gently turned, a germicide should be applied to the conjunctiva. The nitrate of silver (1 or 2 per cent.) solution has stood the test of time. It is used once daily, and need not be neutralized. Of late years protargol in 5- to 40-per-cent. strength solutions has been used by many ophthalmic surgeons. It does not possess any advantage over silver. The presence of a croupous membrane contra-indicates the use of silver. In such cases peroxid of hydrogen should be applied. In the last stage of the disease, when the papillary hypertrophies appear, the application of a weak solution of silver (gr. j to ℥j), or the stick of alum, will be in order.

The treatment of corneal ulceration is preventive and curative. The former includes canthotomy to relieve pressure on the globe, incision of chemosis to prevent strangulation of the minute vessels which ramify in the periphery of the cornea, and frequent cleansing of the conjunctiva to prevent accumulation of pus and infection of the cornea. The curative measures are the use of atropin (gr. iv to ℥j) thrice daily and the frequent washing of the ulcer with formalin (1 to 2000). The latter is the only antiseptic having the property of penetrating tissues. It is the aqueous solution of a gas (hence the bottle must be well stoppered), and is of great

PLATE X.

External Diseases of the Eye.

Fig. 1.
Burn of Conjunctiva and Cornea.

Fig. 3.
Pannus.

Fig. 3.
Essential Shrinking of the Conjunctiva. (Oliver.)

Fig. 4.
Phlyctenular Conjunctivitis and Keratitis.

Fig. 5.
Ophthalmia Neonatorum.

Fig. 6.
Episcleritis.

Fig. 7.
Ulcer of the Cornea.

Fig. 8.
Fascicular Keratitis.

PLATE 10

value in the treatment of corneal ulceration. To prevent prolapse of the iris, in case a marginal ulcer shows impending perforation, eserin ($^1/_{10}$ to $^1/_8$ of 1 per cent.) is to be used, provided iritis is not present. Unfortunately this drug increases iris-hyperemia, and cannot be employed in iritis.

Ophthalmoblennorrhea Neonatorum.—This is an acute inflammatory conjunctivitis occurring in the newborn, and is due to the gonococcus.

SYMPTOMS.—Usually on the second or third, less often on the fourth or fifth, day the infant shows a redness of the eyelids with a muco-purulent discharge. Slight redness of the conjunctiva is succeeded by great swelling of the lids, chemosis, and abundant purulent discharge (Fig. 5, Plate X). Generally one eye is infected first, and if promptly treated the process is much less severe in the second eye. The symptoms are the same as in gonorrheal ophthalmia of the adult, although in the case of the infant the process is less severe and corneal complications are less frequent. The chief danger is to the cornea. There is great swelling of the retrotarsal fold, and when the upper lid is everted the fornix comes into view as a red, rough, suppurating mass resembling granulations. In some cases, where the swelling of this fold is great, the upper lid becomes everted spontaneously when the child cries.

ETIOLOGY.—The disease is due to inoculation by the gonococcus either while the child is passing through the maternal passage or shortly thereafter. In a few cases it is evident that the infection must have occurred *in utero*, since the disease was present at birth. Strzeminski believes that the gonococcus can penetrate the intact fetal membranes. Infection is common in slow labors and in face presentations. Vaginal secretion, which is adherent to the eyelashes and lid-margins, may gain access to the conjunctiva when first the child begins to open and close its eyes. Rough attempts at washing may introduce the infection into the *cul-de-sac*. There are cases of purulent ophthalmia in the newborn which are due to infection by bacteria other than the gonococcus, as will be explained later on.

DIAGNOSIS.—This should present no difficulty. Any redness or discharge about the eyes of an infant, occurring during the first week of life, is to be regarded as gonococcic infection unless bacteriologic examination shows the contrary.

PROGNOSIS.—While a serious disease, when recognized early and properly treated few cases end in loss of the eye, although often the cornea is left with a small cloud. If the case is seen while the cornea is clear the prognosis is favorable, except in diphtheritic types or in those of general malnutrition. If not treated promptly and properly, 80 per cent. of these cases will end in blindness, owing to the sloughing of the cornea. At the first visit the surgeon should examine the cornea, since the prognosis depends on its condition.

PROPHYLAXIS.—In no other disease have prophylactic measures met with such brilliant results. Vaginal antiseptics should be used before birth, and immediately after delivery the child's eyes are to be washed with

bichlorid solution (1 to 8000). Then a drop of 2-per-cent. strength solution of silver nitrate is to be placed on each cornea. This causes considerable reaction, but prevents the disease. In three instances fatal bleeding from the conjunctiva has followed the prophylactic application of silver. The formation of small corneal opacities is of more frequent occurrence. The method described is particularly valuable in hospital practice and in private families where gonorrheal infection of the mother is suspected. A less efficient method of prophylaxis is the cleansing of the lids with bichlorid wash and the irrigation of the conjunctiva with 5-per-cent. boric acid solution. Other prophylactic applications are protargol (20 per cent.) and perchlorid of mercury (1 to 3000). So important is prophylaxis by silver, which is known as Credé's method, that in some foreign countries its practice is made obligatory by law. More than 30 per cent. of the inmates of German and Austrian asylums for the blind were rendered sightless by ophthalmia neonatorum. Fuchs states that, of the 300,000 blind persons in Europe, 10 per cent. may be attributed to this disease.

TREATMENT.—The same principles which govern the surgeon in the treatment of gonorrheal ophthalmia in the adult will obtain in this disease. Cleanliness is to be strictly observed. When suppuration begins, the everted lids are to be painted once daily with a 2-per-cent. strength solution of nitrate of silver. Where the granular masses are abundant, stronger solutions (4 to 8 per cent.) can be used until the flow of pus becomes less; then the weaker solution is used until the child is well. Strong solutions of silver must not be brought into contact with the cornea. Toward the latter part of the treatment a weaker solution (gr. ss to ℥j) is to be used. Corneal complications are to be treated as in the adult, except that the atropin solution must be a weak one (gr. $^1/_4$-j to ℥j).

Non-gonorrheal Purulent Conjunctivitis.—Under this head it is proposed to describe briefly those cases of purulent ophthalmia presenting the clinical signs of gonococcic infection, in which the gonococcus is shown by bacteriologic examination to be absent. The importance of a demonstration of the presence or absence of the gonococcus is unappreciated by many surgeons; yet it has a direct bearing on questions of veracity and on the treatment. Grandclément states that if strong solutions of silver are used in non-gonorrheal purulent conjunctivitis the danger of corneal suppuration is increased. Myles Standish insists that in all cases of purulent conjunctivitis the diagnosis should depend on the bacteriologic findings, not on the clinical appearance. He states that ulcers of the cornea with very rapid necrosis may be due to the Klebs-Loeffler bacillus. In such cases diphtheria antitoxin gives favorable results as regards both the conjunctivitis and the corneal complication. Francisco, in a study of 40 cases of ophthalmia neonatorum, found the gonococcus in 30, and in several of the remaining cases the Koch-Weeks bacillus was present. The average duration of the gonococcic cases was 53 days; of the non-gonococcic cases, 36 days. Parinaud mentions cases of conjunctivitis in the newborn in which there is

little pus, much lacrimation, and moderate palpebral injection. In these cases the pneumococcus is present. Groenouw found the bacillus coli communis in 6 of 40 cases of ophthalmia neonatorum, and Bietti has reported a case in which the colon bacillus was the only microörganism found. Haglund found the diplococcus intracellularis meningitidis in the conjunctival secretion of a boy whose eye was lost by a purulent inflammation. Recently, in St. Louis, several cases of purulent ophthalmia in babes have been shown to be due to the bacillus coli communis.

Treatment.—In cases of non-gonorrheal purulent conjunctivitis the treatment will include cleanliness, the use of antiseptics, and attention to the general condition of the patient. If the Klebs-Loeffler bacillus is present the antitoxin of diphtheria is to be employed.

Croupous Conjunctivitis (Pseudomembranous, Plastic, or Membranous Conjunctivitis). — This disease is characterized by the formation of an exudate on the conjunctiva, where it coagulates and forms a membrane.

Etiology.—Croupous conjunctivitis is the result of many different causes, the chief of which are bacterial, mechanical, or chemical. Among the bacterial causes are the Klebs-Loeffler bacillus, the streptococcus, the pneumococcus, the bacillus of Weeks, the gonococcus, and the xerosis bacillus.

Loss of substance of the conjunctiva is followed by a fibrinous deposit under which healing progresses. This process is seen after wounds—as, for example, after tenotomy—and after injuries. A formation of false membrane follows the application to the conjunctiva of an irritant which may be organic (infusion of jequirity) or inorganic (ammonia, nitrate of silver in strong solutions). By painting the conjunctiva with ammonia Sourdille was able to produce at will either a croupous or a diphtheritic form of conjunctivitis, according as the strength of the irritant was varied. The same result can be had by the application of jequirity. There is a relationship between croupous conjunctivitis and scrofula and eczema. Age is an etiologic factor, the disease having not been observed in the newborn and rarely among adults, while it is found in the majority of cases between the sixth month and seventh year.

The relation of croupous to diphtheritic conjunctivitis has been the cause of much controversy. At the present time the views of Coppez, who finds that the symptoms, both local and general, gradually merge in the two affections, are accepted. Some cases diagnosticated as croupous conjunctivitis have been followed by fatal systemic infection, and others by post-diphtheritic paralysis. The croupous deposit on the conjunctiva may be present coincident with diphtheria of the throat, and in some cases of croupous conjunctivitis the true diphtheria bacillus has been found. Coppez's view of the identity of these affections has been strengthened by therapeutic tests. McQueen, in a case where the clinical diagnosis was croupous conjunctivitis, and the membranes which re-formed after removal were found to contain diphtheria bacilli, injected antidiphtheritic

serum with success. Gossetti and Iona found the diphtheria bacillus present in 6 of 29 cases of croupous conjunctivitis. While the last word has not been said concerning the relationship of these types of conjunctival inflammation, it is necessary to distinguish clinically between the croupous and diphtheritic forms.

SYMPTOMS.—Croupous conjunctivitis presents itself as a mild or as a severe type. The mild form begins as an ordinary conjunctivitis followed by swelling of the lids, which are soft, pliable, and generally are not painful to the touch. The characteristic false membrane follows in a few days. It is formed in the fornices and tarsal conjunctiva, the bulbar portion of the membrane being exempt. The deposit is of a grayish-white, translucent, porcelain-like appearance. When stripped off, it leaves a raw and perhaps a bleeding surface: a diagnostic point which serves to distinguish it from the diphtheritic form. The false membrane generally disappears in about two weeks. The signs of an ordinary conjunctivitis reappear and regeneration becomes complete. The cornea is rarely involved.

A recurring form has been described in which the membrane is formed again and again, the process lasting for months or longer.

The severe type of the disease, caused by streptococcic infection, shows great swelling of the lids, considerable discharge, and rapid destruction of the cornea due to the spreading of the exudation upon the bulbar conjunctiva.

DIAGNOSIS.—Croupous conjunctivitis may be confounded with diphtheritic and gonorrheal inflammations. In diphtheritic conjunctivitis the exudation, instead of being limited to the surface, involves the deeper layers of the conjunctiva. The bulbar portion of this membrane is involved and corneal ulceration is frequent. In gonorrheal conjunctivitis the discharge is much more purulent than in the croupous form. Croupous conjunctivitis is never found among the newborn. As a matter of fact, the diagnosis cannot be made in all cases by the clinical signs. Dependence can be placed alone upon the bacteriologic findings.

TREATMENT.—The sound eye should be protected by a bandage or by a Buller shield. Nitrate of silver, protargol, and other irritants are positively harmful in croupous conjunctivitis. Their use is not to be advised as long as the pseudomembrane is present. The local application of iced compresses, or, if the patient is feeble, the employment of hot applications, will be beneficial. The conjunctiva is to be washed three times a day with bichlorid of mercury (1 to 5000), boric acid, or a normal salt solution. Some surgeons report good results from sprinkling sulphate of quinin on the conjunctiva, and others use this remedy in the form of a lotion. After catarrhal symptoms have become established, weak solutions of silver can be applied cautiously. If the cornea is involved a bacteriologic examination should be made. If the Klebs-Loeffler bacillus is found, diphtheria antitoxin should be used. Aristol has been recommended as a local application in croupous conjunctivitis with corneal ulceration.

Diphtheritic Conjunctivitis, which is rarely seen in the United States and in England, but is common in Germany and in some other Continental countries, is an extremely serious disease. It is a specific inflammation characterized by the formation of an exudate on and within the layers of the bulbar and tarsal conjunctiva.

SYMPTOMS.—Clinicians distinguish three stages: those of infiltration, suppuration, and cicatrization. In the stage of infiltration, which lasts from six to ten days, the initial symptoms are similar to those attending purulent ophthalmia, with the addition that pain is more severe in diphtheritic conjunctivitis. The lids are stiff and boardlike. The conjunctiva is of a gray or buff color, and is covered with an exudation. If this is stripped off, the surface beneath is of the same color, not red and bleeding as in croupous conjunctivitis. The bloodless condition of the conjunctiva, which is produced by coagulation of an exudate compressing the vessels, tends to produce necrosis, and this destructive process may advance so rapidly that the eye may be destroyed within twenty-four hours. There is danger of corneal necrosis at any time during the stage of infiltration. Patches of membrane are often found in the throat, and any excoriated spots about the cheeks, nose, or mouth readily become infected when brought into contact with the secretions.

The stage of infiltration is succeeded gradually by that of suppuration. The lids become softer, the fibrinous exudate disappears, and the conjunctiva begins to look reddish, raw, and succulent. A purulent discharge is present. This stage ends and is followed by that of cicatrization. This leaves the conjunctiva atrophic and shrunken, with frequently great deformity of the lids. Thus, trichiasis, entropion, symblepharon, and even exophthalmos may be produced. Involvement of the cornea, which is a frequent complication, assumes the form either of local ulceration or of diffuse infiltration. It is always secondary, there being no such process as primary diphtheria of the cornea. Coppez states that cases reported to have been primary diphtheria of the cornea were principally instances of corneal diseases from other causes in which the finding of the xerosis bacillus led to an incorrect diagnosis. The advent of corneal involvement causes great pain, and, as a rule, the process occurs early in the case.

ETIOLOGY.—Diphtheritic conjunctivitis is due to the Loeffler bacillus (Fig. 2, Plate IX). The disease occurs chiefly in children between the ages of two and eight years. Generally both eyes are involved. It may occur primarily, or may be secondary to nasal or faucial diphtheria.

DIAGNOSIS.—The chief clinical signs of croupous and diphtheritic conjunctivitis having been enumerated, it remains to be said that the diagnosis may be impossible without bacteriologic examination. Coppez states that there is a group of bacteria (the xerosis bacillus, the pseudodiphtheria bacillus of Hoffmann, the bacterium septatum of Gelpke) which can be distinguished from the true diphtheria bacillus only by culture or by inoculation experiments. To make certain of the diagnosis of ocular diph-

theria he advises examination of cover-glass preparations, cultures on serum-agar, and the use of the Ernest-Neisser double coloration. A competent bacteriologist can make these examinations and report the result the first day the case is seen. Owing to the general presence of the xerosis bacillus in the conjunctiva, the making of a microscopic examination of the discharge is less valuable here than in faucial diphtheria.

TREATMENT.—In this disease both general and local treatment must be employed. Immediately the case is recognized as one of diphtheritic conjunctivitis, an injection of antitoxin must be given. The earlier this treatment is instituted, the less will be the danger of corneal sloughing. The injection should be repeated at the end of twenty-four hours. Improvement should be noticeable at the end of a few hours. If the antitoxin is used later, the beneficial effect will be slow in appearing. When the antitoxin treatment is begun early the cornea can be saved in nearly all cases.

Locally mild antiseptic washes are to be used. To protect the cornea from friction it will be advisable to place vaselin in the *cul-de-sac* twice daily. The sound eye should be covered with a Buller shield.

Phlyctenular Conjunctivitis (Conjunctivitis Eczematosa; Scrofulous, Lymphatic, or Strumous Ophthalmia; Phlyctenular Ophthalmia).—This is a form of conjunctival and corneal inflammation occurring chiefly in children and characterized by eruption of phlyctenulæ (Fig. 4, Plate X). These are accumulations of lymphoid cells, forming red elevations about the size of a millet-seed and situated upon the limbus of the conjunctiva. Soon the epithelium at the apex of the phlyctena separates, the underlying tissue becomes softened and breaks down, and thus a minute gray ulcer is produced. Often the same process occurs simultaneously in the cornea. For this reason some ophthalmic writers have considered the disease in its entirety under one heading, devoting a separate chapter to it. The author will treat of phlyctenular conjunctivitis in this place, and of phlyctenular keratitis in the succeeding chapter, it being understood that no sharp line of demarcation can be drawn between these subjects.

ETIOLOGY. — There is no general agreement as to the etiology of phlyctenular conjunctivitis and keratitis. Horner regarded the phlyctenulæ as merely ocular manifestations of eczema, and this view has been confirmed recently by the studies of Sydney Stephenson. He found, among 669 cases of phlyctenular affections of the eye, that in 355 (53.06 per cent.) eczema was present on examination, had been present previously, or is known to have appeared later. He demonstrated eczematous changes in the skin, in the nasal and buccal mucous membranes, and in the external auditory meatus. The phlyctenular diseases of the eye go hand in hand with tuberculous manifestations, such as swelling or abscess of the glands below the jaw, joint and bone lesions, otorrhea, dactylitis, pulmonary phthisis, and scrofulodermata. The occasional observation of phlyctenular conjunctivitis and keratitis in apparently healthy individuals may be ex-

plained, as Gradle states, by the assumption that a small tuberculous focus has been overlooked. Many writers assume that the active microörganism is the staphylococcus pyogenes aureus or albus, which is present under the affected epithelium. Phlyctenular ocular disease often follows measles or scarlatina. Rhinitis is always present, adenoid vegetations are common, and digestive disturbances often exist in these patients. In some cases hereditary syphilis is a factor. The disease is found chiefly between the fourth and fourteenth years, although it is occasionally seen in adults.

Symptoms.—The subjective symptoms are pain, photophobia, lacrimation, and blepharospasm. The child avoids the light, hides in a dark corner, or buries its head in a pillow. Examination is difficult, owing to its struggles, and separation of the lids requires retractors. The child often presents a characteristic strumous appearance, being pale and thin, or flabby. Enlarged cervical lymphatic glands, adenoid vegetations in the pharynx, thick lips, eczema of the nose and upper lip, and purulent otitis media are often seen in these children.

Inspection early in the case shows vesicles which form flattened prominences, measuring from 1 to 4 millimetres in diameter. They are of a grayish-red color and are surrounded by reddened conjunctiva. A few days later, the epithelium having been cast off, ulcers (phlyctenular ulcers) appear. These are smaller than the vesicles, because of a growth of epithelium from the periphery. The disease may present one or several phlyctenulæ. After a variable time these heal and leave no scar on the conjunctiva. When appearing on the cornea a faint scar or deep cloud may remain, or the ulcer may perforate. In the multiple form the symptoms are generally much more complicated and relapses are common. The disease is not contagious. When a phlyctenular ulcer advances from the periphery toward the centre of the cornea, it is followed by a narrow band of vessels. The name *fascicular keratitis* (Fig. 8, Plate X) has been given to this condition. In phlyctenular conjunctivitis there is abundant lacrimation with little or no discharge. Eczema and blepharitis marginalis are often present. The life period of each phlyctena is from a few days to several weeks.

Diagnosis.—Attention to what has been mentioned will prevent errors in diagnosis. In herpes of the conjunctiva, with which disease phlyctenular conjunctivitis may be confounded, the vesicles are transparent, appear in clusters, and do not show a predilection for the limbus. Furthermore, they are more transient. In spring catarrh the elevations are larger and do not ulcerate. Trachoma of the bulbar conjunctiva is associated with the same process in the conjunctiva of the lids and rarely involves the limbus.

Prognosis.—Generally this is favorable, the disease yielding promptly to proper treatment. When the cornea is deeply involved scars will be left. A small number of cases will prove refractory to the most careful treatment. Localized infiltrates, not the phlyctenulæ, are referred to here, the latter being transient lesions.

TREATMENT.—Recognizing that phlyctenular conjunctivitis is largely the local expression of a systemic disturbance, it will be the surgeon's duty to find the cause and remove it, while at the same time local treatment is to be employed. Errors of diet must be corrected, and it is well to exclude the sugars and starches, feeding the child on meats, milk, and oatmeal. The meals should be given with regularity, and eating between them or the use of miscellaneous articles of diet must be forbidden. The child with phlyctenular conjunctivitis or keratitis should not be kept indoors or in a darkened room. On the contrary, he should be compelled to obtain fresh air and sunlight. The eyes should not be bandaged. The skin should be kept clean and the bowels open. Often the use of calomel internally will improve the health. The palatable preparations of iron, codliver-oil, and of quinin can often be employed with benefit. The syrup of the iodid of iron, 20 or 30 drops three times a day after meals, is an accepted remedy. In those cases which do not respond to the general treatment just mentioned, and the local measures to be described, the salicylate of soda should be used internally in large doses (gr. iij every four hours, for a child of five years). The effects must be carefully watched, depression and tinnitus being signals for the temporary interruption of the doses. If this treatment is not followed by improvement after a few days, it will be useless to continue it (Gradle).

Locally the use of atropin is indicated. Where there is much irritation holocain can be used occasionally. If, as is generally the case, the child comes to the surgeon after the vesicles have burst, mercury should be used locally. There is probably no mercurial preparation more efficient than a properly prepared ointment of the yellow oxid (gr. j to 3j). A small piece of this remedy is to be placed under the upper lid, once a day, by means of a probe. The eye is then to be massaged. Finely powdered calomel dusted on to the ulcers is an appropriate application. It should not be used if the patient is taking iodid of potassium internally, since the potassium iodid in the tears, uniting with the calomel, will form double iodids and cause irritation. The treatment with the yellow oxid must be continued for ten days after all signs of redness have disappeared. The patient's refraction should then be examined. In addition to the treatment outlined above it will be necessary to give attention to the concomitant lesions of the nose, ear, skin, pharynx, and lymphatic glands. In other words, the patient should be generally overhauled and all pathologic conditions should be treated.

Herpes of the Conjunctiva (Herpes Simplex).—Herpes of the conjunctiva, occurring apart from herpes zoster ophthalmicus, is a very rare affection. In it vesicles are found on the bulbar conjunctiva between the limbus and canthus. They appear in clusters and often the cornea is involved simultaneously. The disease occurs chiefly in adults. Photophobia and abundant lacrimation are marked symptoms. The intact vesicles are rarely seen, because they rupture early. They leave small ulcers to which

shreds of epithelium are attached. The disease is to be distinguished from phlyctenular conjunctivitis by the description given above. It is to be treated by attention to the general health and by the use of the yellow oxid ointment.

Papular Conjunctivitis.—A few cases have been recorded of papular conjunctivitis. Schreiber has described the case of a man, aged twenty-one years, who presented eroded papules on the genitals, and later similar papules appeared on the bulbar conjunctiva. The condition was considered phlyctenular until the presence of a peculiar efflorescence led to the employment of specific treatment, under which the disease rapidly disappeared.

Pemphigus of the Conjunctiva.—This rare disease, which was observed by Horner 3 times in 70,000 eye cases, is characterized by the development of successive crops of bullæ. Pain, photophobia, and lacrimation are prominent symptoms. The disease is generally associated with pemphigus of other parts of the body, such as the nose, mouth or throat, or skin. The bullæ, which begin in the fornix or bulbar conjunctiva, rupture and leave grayish, ulcerated areas. These slowly cicatrize, causing shrinking and deformity. Meanwhile other bullæ appear. Thus, the process continues for months or years. The conjunctiva becomes dry, meridional bands pass between the lids and the globe forming the condition known as symblepharon posterius, and in severe cases there is complete obliteration of the *cul-de-sac* (total symblepharon). Ulcers appear on the cornea, which becomes opaque and staphylomatous. Thus, vision is lost. The cause of the disease is unknown, and treatment is without avail. Arsenic may be given internally. *Essential atrophy of the conjunctiva* (Fig. 3, Plate X) seems to be due to pemphigus, although some writers describe it as an independent disease.

Parinaud's Conjunctivitis (Lymphoma of the Conjunctiva).—This is a rare and peculiar form of conjunctival disease which has been studied by Parinaud, Gifford, Goldzieher, Despagnet, and Dominique. It is characterized by the development of gigantic lymph-follicles in the conjunctiva and by the presence of lymphomata in the neck on the same side as the diseased eye. The infection is supposed to be of animal origin, but this has not been proven and a specific microörganism has not been demonstrated. The disease begins suddenly with great thickening of the lids. There is a muco-purulent discharge, which may be abundant or scanty. Within the first week or two, large, polypoid, pedunculated granulations appear on the tarsi, in the fornices, on the ocular conjunctiva, or in all these places. Coetaneous with the conjunctivitis, the pre-auricular and retromaxillary glands swell and in some cases the cervical and submaxillary glands are similarly affected. The swelling may attain enormous proportions and suppuration frequently occurs. There are rigors and a rise of temperature. With one exception, in all the cases so far recorded, the disease has been unilateral. Spontaneous cure occurs in from two to six months, the disease being rebellious to treatment. The visible granulations, which at first

are red or grayish red, often hide smaller ones which are yellowish. They may become eroded. The prognosis seems to be favorable, since in only one of the reported cases has the cornea been involved.

As regards treatment, Parinaud uses nitrate of silver, Abadie favors the galvanocautery, Gifford thinks that copper sulphate did good in a severe case which he treated, and other ophthalmologists have used iodoform ointment. Ablation of the granular masses has been recommended.

Lithiasis of the Conjunctiva (Uratic Conjunctivitis).—This disease is characterized by a deposit of crystals of uric acid or sodium urate, in the acini of the Meibomian glands. The disease is associated with the gouty or rheumatic diathesis. Patients with lithiasis complain of a pricking sensation in the eyes and the feeling of a foreign body under the lids. Examination shows the deposits existing as numerous small concretions of a yellowish-white color. Both the palpebral and bulbar portions of the conjunctiva are hyperemic and the anterior scleral vessels are engorged. The disease is more common in elderly than in young subjects. It is proper to state that Herbert, who has carefully studied the conjunctival changes produced by chronic inflammation, considers "lithiasis," "infarcts of the Meibomian glands," and "mycosis" as cyst-formations sequent to the closing of epithelial tubules. The downgrowth of these tubules is found not only in papillary trachoma, but also in all forms of chronic conjunctivitis. The cheesy material found in these cysts results from the accumulation and degeneration of epithelial and wandering cells. The treatment consists in removing the crystallized masses with a cataract-needle under cocain anesthesia. This treatment is to be followed by the use of a boric acid wash locally and the internal administration of the salts of lithia. Attention to the general health is required in these cases. The disease is prone to recurrence.

Egyptian and Military Conjunctivitis are terms which should be excluded from ophthalmic nomenclature, since they have been applied indiscriminately to several different affections, among which are the gonorrheal, acute catarrhal, and trachomatous forms of conjunctivitis.

Variolar Conjunctivitis.—Hyperemia of the conjunctiva usually accompanies variola. About the fifth day of the eruption the conjunctiva may show a catarrhal form of inflammation which readily yields to simple treatment. The intensity of the conjunctivitis is in proportion to the involvement of the face and eyelids. Pustules rarely form upon the conjunctiva. When they occur they resemble phlyctenulæ. A favorite site for the pustules is the area between the corneal margin and the inner or outer canthus. Pustules may appear on the limbus, in which event corneal ulceration is likely to supervene. Chance states that pustules may form on the tarsal conjunctiva and on the caruncle, but are never found at the fornix. In the pustular type of variolar conjunctivitis severe inflammatory symptoms, such as chemosis and profuse discharge, are present. Subconjunctival hemorrhages may occur in hemorrhagic variola.

In the treatment of the conjunctivitis of variola mild antiseptic solutions should be used.

Chronic Conjunctivitis is a common disease in the aged, and is characterized chiefly by hyperemia, swelling of the caruncle, hypertrophy of the papillary layer of the conjunctiva, and a scanty muco-purulent discharge. It may be the result of an acute catarrhal conjunctivitis, of an error of refraction, of lacrimal disease, of nasal inflammation, of excessive near work, or of unhygienic surroundings. The treatment includes the removal of the cause, abandonment of injurious habits (smoking, abuse of alcohol, etc.), correction of errors of refraction, and attention to the general health.

Lacrimal Conjunctivitis.—This term is applied to those cases of conjunctival inflammation which are produced by the irritation of the discharge from an inflamed lacrimal sac. Various pathogenic germs are found in dacryocystitis, one of the most important being the streptococcus pyogenes (Fig. 1, Plate IX). From contact with the irritating discharge, the caruncle and plica semilunaris become inflamed. The cause may be easily determined if the surgeon thinks to investigate the condition of the lacrimal apparatus. Often this examination is overlooked and the disease is classed as an intractable chronic catarrhal conjunctivitis. The prognosis will depend on that of the lacrimal disease. The cure of dacryocystitis will cause immediate improvement in the condition of the conjunctiva. If a corneal ulcer has been infected with the lacrimal discharge, the prognosis may be serious, such cases occasionally leading to deep ulceration of the cornea, with perforation and loss of the eye.

Granular Conjunctivitis (Conjunctivitis Granulosa, or Trachomatosa; Egyptian Ophthalmia; Trachoma; Military Ophthalmia; Granular Lids).—This may be defined as a conjunctival inflammation (Fig. 3, Plate VIII) of long duration, characterized by the presence of trachoma bodies or granulations, and ending in cicatricial changes. In this disease, in addition to hyperemia and discharge, there are numerous grayish or pinkish-red bodies, which are about the size of a pinhead. These are particularly abundant in the conjunctiva of the upper lid and in the upper fornix, but also exist in the lower lid and in the bulbar conjunctiva. Trachomatous tissue may appear on the cornea.

Etiology.—The disease originates in infection and produces an infectious purulent secretion. The identity of the microörganism causing it is unknown. Sattler, von Michel, and other ophthalmologists have described a small double coccus which can be cultivated from the contents of a trachoma follicle and which has been named the trachoma-coccus. Inoculation experiments with this coccus have not been productive. Pfeiffer and Ridley have described parasitic protozoa, and Muttermilch has written concerning a fungus named *Microsporon trachomatosum*, with whose pure cultures he claims to have produced trachoma in some of the lower animals. Other microörganisms—gonococci, streptococci, etc.—have been found in the discharge.

The disease is spread by transfer of secretion from a trachomatous eye, the virulence of the process depending upon the quantity and quality of the discharge. The most dangerous epidemics are those characterized by an abundance of yellowish discharge. A trachoma which is kept in check by systematic treatment possesses slight power of contagion. The disease is common in places where many persons are crowded into small quarters and where unhygienic conditions exist, as in barracks, workhouses, orphan asylums, and schools. The agents concerned in passing infectious particles are numerous, and include such media as washing utensils, hands, handkerchiefs, bedlinen, medicine-droppers, etc. In foreign climes, where the heat is great, flies are the chief factors in spreading the contagion. Not age, but race, is an etiologic factor. Thus, trachoma is common among Hebrews, Italians, Egyptians, and other inhabitants of the East. The negro is almost exempt. The disease is common in Ireland. The geographic distribution of trachoma is interesting. It is frequent in Arabia, Egypt, the eastern part of Europe, and the lowlands. Elevated areas, such as the Tyrol, Switzerland, and isolated mountain districts, where the altitude exceeds 6000 feet, are practically exempt. It is also of rare occurrence in Scandinavia and in southern California. The disease is common in the western prairie districts of the United States, particularly in regions where sandstorms are frequent. Ziem states that the prevalence of the disease has kept pace with the destruction of forests and the consequent production of dust and sandstorms. It is supposed by Kuhnt that nasal disease is an etiologic factor. It has been erroneously believed that trachoma was first introduced into Europe by Napoleon's soldiers returning from Egypt in 1798. It is probable that eyestrain, by causing conjunctival hyperemia, predisposes to the development of trachoma. The question whether malaria influences it is unsettled. Persons of the so-called scrofulous temperament are prone to the disease.

FORMS.—The subject of trachoma may fitly be considered under the following subdivisions: 1. Papillary trachoma. 2. Granular or chronic trachoma. 3. Mixed trachoma.

1. The term papillary trachoma (papillary granulation or hypertrophy) means that the papillæ of the upper lid, more rarely of the lower, are enlarged and recognizable by the unaided eye. In mild cases the conjunctiva resembles sandpaper, while in severe ones it has been compared to a pile of velvet. It looks moist, red, and velvety. Early in the case lacrimation is profuse. Later a muco-purulent or purulent discharge appears. The conjunctiva is injected, the papillæ are enlarged, and the characteristic granulations are scattered through the membrane. After a variable period the disease subsides. In favorable cases a cure results, the granulations being absorbed and a smooth conjunctiva remaining. In other patients there are indications of scar-tissue in the mucous membrane, or the disease may pass into the chronic form.

2. Granular or chronic trachoma generally is chronic from the begin-

ning, but in some cases it results from the imperfect disappearance of the papillary form. The characteristic feature is the presence of the trachoma granules. These are round, opaque bodies, of grayish-white color, measuring about two millimetres in diameter. They are deeply set in the conjunctiva and are often confluent, thus forming areas of trachomatous material. They may be scattered over the whole conjunctiva, but are particularly numerous where the adenoid tissue is abundant: *i.e.*, in the fornices, especially the upper one. The conjunctiva may be hyperemic or anemic. There is generally a slight muco-purulent discharge. The eyes burn and smart, near work becoming difficult, particularly at night. The disease may continue thus insidiously for many months, when suddenly an attack of acute inflammation ensues and the picture changes to that of acute inflammatory

Fig. 211.—Trachoma follicles. (Author.)
(Photomicrograph by Dr. H. P. Wells.)

trachoma. Redness, pain, lacrimation, photophobia, discharge, blepharospasm, corneal inflammation and ulceration, and rapid loss of vision are now important symptoms. Acute inflammatory trachoma is simply trachoma to which an acute conjunctivitis has been added.

When, in the quiet form, the lids are everted, the characteristic granulations appear as sago-like elevations which are arranged in rows and are particularly prominent in the fornices. A few isolated trachoma bodies may be seen in the bulbar conjunctiva. The membrane is rough, and does not show the velvety appearance found in the papillary variety. After the advent of inflammatory symptoms the membrane shows swelling of the papillæ, which may obscure the granulations. They may be absorbed, but generally new trachoma bodies appear, and thus the disease continues indefinitely. After a variable period grayish-white bands of connective tissue

are to be seen, marking the appearance of the stage of cicatrization. Thus the disease improves at the expense of the normal conjunctiva, which is replaced by scar-tissue. In this stage the tarsal plates become deformed, producing trichiasis, entropion, and corneal ulceration. The conjunctiva is converted into a pale, bluish-white, atrophic membrane, and the fornices are much reduced in size or are entirely obliterated.

3. Mixed trachoma is a common condition, the papillary and granular forms being present simultaneously. The papillary form involves chiefly the tarsal conjunctiva, while the granules develop luxuriantly in the fornices.

COMPLICATIONS AND SEQUELÆ.—The chief complications of trachoma are corneal ulcers and pannus. *Corneal ulcers* may appear at a place where the cornea is normal or in connection with pannus. The nature of these ulcerations does not demand special attention in this place. *Pannus* (Fig. 2, Plate X) consists in the formation of a new growth of connective tissue which is situated between the epithelium and Bowman's membrane and is provided with blood-vessels. This new tissue presses its way from the periphery toward the centre, and corresponds chiefly to that part of the cornea in relationship with the upper lid. Pannus may disappear entirely, or the enlarged vessels may shrink, leaving small radiating lines to mark the spot where they existed. If ulceration is associated with pannus, the cornea will likely remain clouded in its upper part. Buller has seen cases of trachoma associated with ichthyosis.

Among the sequelæ are trichiasis and entropion. Normally the margin of the lid, at the point where the conjunctiva and skin unite, forms a right angle. In trachoma it is worn away, and the eyelashes, being given a wrong direction *(trichiasis)*, rub on the cornea, producing constant irritation. The contraction of the newly formed connective tissue leads to thickening and incurvation of the tarsal plate (*organic entropion*, Fig. 6, Plate VII) with trichiasis. Continuance of either condition leads to corneal inflammation and ulceration. The lower eyelid often turns outward as a result of trachoma. The thickened conjunctiva presses the lid-margin away from the globe and contraction of fibres of the orbicularis muscle completes the eversion (*spastic ectropion*, Fig. 5, Plate VII). When atrophy of the conjunctiva has become marked, the folds in the fornices disappear and the mucous lining of the lids passes directly into that of the globe *(posterior symblepharon)*. In excessive atrophy the conjunctiva is converted into a dry, tough membrane *(xerosis)*; the cornea suffers at the same time, becoming cicatricial and opaque. In the course of trachoma in the Malay race, and also among the Chinese, Steiner observed *pigmentation of the conjunctiva* in the form of irregular dots and lines. The spots, which are found most frequently on the upper lid, are black. They are without pathologic significance. Among the most important sequelæ are the corneal changes, which include: (1) the formation of scars, following ulceration, by the transformation of pannus into connective tissue; and (2) the

terations in curvature, which may be slight or may produce a decided bulging of the membrane (ectasia of the cornea). Thus it is seen that trachoma, by reason of its complications and sequelæ, is one of the most important of ocular diseases.

PATHOLOGY.—As regards the nature and origin of the changes found in trachoma there is much dispute. Iwanoff, Berlin, and others contend that there is a new product, while Sattler, Raehlmann, and Vincentiis believe that there is simply a change in the normal tissue. Burnett states that the process is probably a combination of both. Attempts which have been made to distinguish histologically between follicular conjunctivitis and trachoma have not been successful. Herbert believes that the difference is in the amount of material present in the two types. He states that in follicular conjunctivitis and trachoma, in addition to an increase in the number of large plasma-cells (rounded cells modified from connective-tissue cells in chronic inflammation) which are distributed through the conjunctiva, there is an hypertrophy of existing follicles and lymphoid tissue, with a new formation of these structures. The changes in trachoma begin in the lymphatic spaces of the lymph-vessels. Such a collection of cells, encapsuled by vessel-walls, by growth gives origin to a follicle. Commonly the follicles are formed by the continued addition of cells grouped in lymph-spaces and lymph-vessels. Absorption of follicles, according to the same authority, takes place in various ways: 1. The cells are carried away in the lymphatics. 2. Other cells become ameboid and travel through the surface epithelium or into the blood-vessels. 3. Others undergo vacuolar or hyalin degeneration. The trachomatous process involves all parts of the conjunctiva, except the ocular portion near the cornea and a narrow strip, three millimetres broad, next to the lid-borders. These parts normally are covered by stratified squamous epithelium. As regards the hypertrophic changes, there is an extension of surface by the formation of papillæ and an increase of epithelium in epithelial downgrowths. These epithelial tubules often lead to the formation of cysts possessing yellowish and cheesy contents. In the stage of atrophy there is a disappearance of areas of the normal fibrous and elastic matrix of the conjunctiva, with the formation of scar-tissue. The development of papillæ is not characteristic of trachoma, since they may attend any type of conjunctivitis of considerable duration.

DIAGNOSIS.—The diagnosis of trachoma generally presents little difficulty. Such diseases as chronic catarrhal conjunctivitis, vernal catarrh, and the papilliform swellings of purulent conjunctivitis have been sufficiently described and will not embarrass the careful observer. Between the false, or follicular, granulation and the true, or "sago-grain," granulation differentiation may sometimes be difficult. The chief characteristics of these affections, which are different clinically, but not histologically, are given by Stephenson as follows:—

Follicular, or False, Granulation.	"Sago-grain," or True, Granulation.
1. Oval or roundish, transparent bodies, the diameter of which seldom or never exceeds 1 or 1.5 millimetres. They often possess a faint-yellowish hue, and are usually arranged in rows. Their tendency is to remain discrete: that is, separate from one another. They are always larger in the inferior fornix.	1. Round, opaque, ill-defined bodies of grayish-white color and extreme friability. Firmly and deeply imbedded in the conjunctiva, their diameter often reaching 2 millimetres or more. They tend to become confluent, thus forming areas of trachomatous material. They are always larger and more numerous in the upper fornix.
2. Seldom are associated with much change in the structure of the conjunctiva.	2. Structural changes are always present in the conjunctiva.
3. Papillary hypertrophy of the upper lid is trivial.	3. Papillary hypertrophy of the upper lid is marked in many of the cases.
4. The tarsus is not implicated.	4. The tarsus is often involved.
5. The growths disappear spontaneously without forming scar-tissue.	5. Spontaneous cure occurs only with the onset of scarring, which may be slight or extensive according to the degree of development of the original granulations.
6. No drooping of the upper lid.	6. Upper lid droops in most cases.
7. Pannus and corneal ulcers are absent.	7. Pannus and corneal ulcers occur in at least 25 per cent. of the cases.
8. Trichiasis, entropion, and shrinking of the *cul-de-sac* do not occur.	8. Trichiasis, entropion, and shrinking of the *cul-de-sac* occur very frequently.
9. Occurs chiefly in persons under twenty years of age.	9. May occur at any age.
10. Not contagious.	10. Conditionally contagious.

Prognosis.—When seen early and treated persistently the prognosis of trachoma is favorable. Corneal ulceration and pannus often give better results than would be expected, although vision is often reduced. Trichiasis and entropion can be relieved by operative treatment. In the stage of atrophy treatment is of little use. Unfortunately persons with trachoma often cease treatment too soon, and under such circumstances they are sure to suffer a recurrence. Such cases often end in blindness or in serious reduction of vision. A trachomatous eye is always liable to attacks of acute inflammation in response to a fresh irruption of trachoma follicles or to external irritants.

Treatment.—The treatment of trachoma is prophylactic, medical, and surgical. When possible these cases should be isolated. In private practice each patient should have his own towels, soap, washbowl, and handkerchiefs. In asylums and schools the lavatory arrangement should include what is known as the "jet system," which makes it impossible for the same water to be used by more than one person. Cleanliness is always in order in trachoma. Hot water and Castile soap are probably as efficient as any of the numerous antiseptic washes.

The medical treatment may be formulated as follows: In acute inflammatory attacks cleanliness, hygienic measures, and atropin are to be employed until the pain and inflammation have subsided. Then once daily the conjunctiva of the lids and fornices is to be painted with a solution of nitrate of silver, of the strength of 2 or 3 grains to the ounce, until the secretion ceases. If the cornea is involved atropin is to be used once or twice a day. This treatment should be continued week after week, but it must not be kept up indefinitely, because of the danger of producing argyrosis. In applying the silver solution the surgeon should see that it reaches the folds in the upper fornix. Caustics are absolutely to be eschewed in this as in every other form of conjunctivitis. What is wanted is stimulation of the absorbents, not destruction of the membrane. In many lands, after the secretion has diminished, a stick of lunar caustic or copper sulphate is employed. Schiele uses the solid stick of iodic acid, and claims that it does not cause a scar. Nesnamoff employs a 1- or 2-per-cent. strength solution of iodin in liquid petrolatum. Seabrook commends this treatment in chronic cases with little secretion. Ichthyol in 10- or 20-per-cent. strength solutions has found favor with some ophthalmic surgeons. In the hands of many practitioners copper and other caustics are positively harmful. When used they are to be employed simply as irritants, not as escharotics, and their application will not be in order until after the discharge has practically ceased. Instead of copper, a stick of alum can be used as a stimulating agent. It is possible, as Thompson has remarked, that trachoma is a much different disease in the Mississippi Valley from that type found in some far-distant place, and that the treatment should vary accordingly. As the case improves under the treatment outlined, the period between the applications can be lengthened and the patient can make use of a collyrium of sulphate of zinc, boric acid, protargol, or argyrol. The use of a 3- to 10-per-cent. strength ointment of citrate of copper in white vaselin is recommended by Wright, of Mobile. He states that it produces absorption of the granulations, is non-irritant, and can be used by the patient.

Under medical treatment improvement of trachoma is often a slow process. Hence various surgical measures have been advocated, and many of these are of great antiquity. Among those worthy of mention are scarification, expression, brossage, grattage, excision of individual granulations, excision of the *cul-de-sac*, the application of the galvanocautery and electrolysis, or the use of the x-rays. These measures, as a rule, are to be employed only in cases in which acute symptoms are absent and which resist medical treatment, although some eminent authorities prefer them to medical measures. The objection to most surgical procedures is that the scar resulting from the operation may more than offset the good derived from the operation. Of the surgical procedures, the least objectionable is expression. This can be done in various ways, but the use of the roller forceps is preferred. The instrument is used to express the contents of

the trachoma follicles, and this is accomplished rapidly and safely under local or general anesthesia. Expression may be used alone or it may be combined with scarification of the conjunctiva and the local use of a germicide which is rubbed into the tissues by means of a stiff brush. The author prefers expression used alone and followed immediately by cold compresses. Later for several weeks a solution of nitrate of silver is to be applied. In using expression care must be taken to press out the trachomatous material in the region of the fornices and canthi. Stephenson, Walsh, and Mayou have recently reported favorably on the treatment of trachoma by x-ray tube exposure.

Pannus generally clears up *pari passu* with the improvement in the conjunctiva, but in some cases, in which there is a formation of dense connective tissue, it may require special treatment, such as the use of jequirity or the performance of periectomy. Jequirity, which was introduced into ophthalmic practice by de Wecker, is employed to set up a violent corneal inflammation in the hope that with its subsidence the pannus will disappear. It is used in an infusion (3 to 5 per cent.) applied to the everted lids, or the powdered drug may be dusted on to the conjunctiva. The latter method is highly recommended by Cheatham. The jequirity treatment must be used with caution, since some cases have been reported in which its employment was followed by destruction of the cornea. In many cases it has a curative effect on pannus.

Landolt and Holmes have reported good results from the treatment of pannus by boric acid and massage. The everted upper lid is dusted with the powder and then the eyeball is massaged with the lid intervening, a local anesthetic having been previously applied.

Since pannus is a corneal complication of a circumcorneal disease, the excision of a piece of conjunctiva adjacent to the cornea has been done for the purpose of producing an area of cicatricial tissue, which shall act as a safeguard against invasion. Boeckmann, who has had an extensive experience with this operation (periectomy), regards it as a harmless and efficient treatment for pannus.

Vaccinia of the Conjunctiva is of rare occurrence, and is characterized by violent redness and swelling of the affected membrane. The infection is carried by unclean fingers and runs the course of vaccinia. It tends to recovery and calls for little treatment.

Varicella of the Conjunctiva has been described by Hilbert.

Conjunctivitis Petrificans (Acute Calcification of the Conjunctiva).—In 1893 Leber described this rare disease, which is characterized by the presence in the conjunctiva of lime in organic, crystallizable combination. The lesion appears as white, opaque spots, with slight or no inflammatory and subjective symptoms. The disease spreads spasmodically, new foci appearing while others are healing, the process lasting for months or years. The smaller foci vanish by absorption, while the larger ones leave shriveled, thickened spots in the conjunctiva. In some cases the disease produces

blindness through corneal involvement. When it is possible to do so, the foci are to be excised.

MISCELLANEOUS DISEASES OF THE CONJUNCTIVA.

Ophthalmia Nodosa (Raupenhaar-ophthalmie) is due to the entrance of caterpillar-hairs into the conjunctiva or cornea and iris. The disease is characterized by intense pain and the appearance of nodules in the tissues involved. Lacrimation and photophobia are present. Violent inflammatory symptoms supervene some weeks or months after the receipt of the injury, and iritis and iridocyclitis are frequent complications. Where nodules are found in the iris the disease may simulate tuberculosis. Often careful and repeated examinations are necessary before the hairs can be found. Excision of the nodules and the continued use of a mydriatic are called for.

Filaria.—In addition to the cysticercus an entozoön known as *Filaria loa* has been observed beneath the conjunctiva. The parasite occurs among the natives of the west coast of Africa and in persons who have visited this region. The worm varies from twenty to seventy millimetres in length and is about one-half to one millimetre broad. It is round, firm, transparent,

Fig. 212.—Conjunctivitis petrificans. (Reif.)

and colorless. By its movements beneath the conjunctiva or under the skin of the eyelids it keeps the eye irritated. The treatment is wholly surgical. The worm should be removed through a conjunctival incision.

Pterygium (Fig. 7, Plate VIII) is a fleshy and vascular growth, triangular in shape, springing from the conjunctiva, generally of the outer or inner canthus, and extending to the cornea, with which it is united. It is frequently located over the course of the internal rectus muscle. The same eye may present two pterygia, one on the nasal, the other on the temporal, side; and the two may meet in the centre of the cornea. In rare instances pterygium is found growing from the centre of the lower or upper eyelid, as in Fig. 213. If of recent origin, the growth will appear reddish and succulent (pterygium crassum); if somewhat old and atrophic, it will appear thin and membranous (pterygium tenue). The disease may remain stationary or may become progressive, rapidly encroaching upon the cornea. In the course of years it may completely cover the cornea and thus do great damage to vision. The conjunctiva forming the pterygium may be tense, and thus the plica semilunaris may be obliterated. Such

growths often cause irritation by the constant tension and may even limit the action of the ocular muscles. A pterygium often will become irritated and inflamed, particularly in the summer, when dust is abundant.

ETIOLOGY.—Pterygium, while generally found in adult and middle life, is occasionally seen in young persons. It is common among farmers, thrashers, ranchmen, teamsters, stonecutters, and other persons who are much exposed to the elements. Heat, a dry atmosphere, high winds, exposure to sunlight, and the irritating effect of alkaline dust are etiologic factors. The disease is much more frequent in the southwestern than in other portions of the United States. It begins either in degenerative processes following on pinguecula—this growth pulling the conjunctiva on to the cornea, where it becomes attached—or it follows upon a small

Fig. 213.—Traumatic pterygium. (OLIVER.)

ulceration at the margin of the cornea. Savage believes that Fuchs's view that pterygium originates from pinguecula is incorrect.

PATHOLOGY.—Histologic examination shows that a pterygium is simply an hypertrophy of the conjunctiva which involves the epithelial and anterior elastic layers of the cornea.

PROGNOSIS.—If the pterygium has encroached upon the pupillary area of the cornea, there will be diminution in vision after the excision of the growth. If the entire cornea is covered, the removal of the pterygium will improve vision, but normal vision should not be expected. In general, it may be said that the prognosis in cases of pterygium is favorable. The condition may return to a less extent than before operation.

TREATMENT.—Medicines are useless in the treatment of pterygium; operation alone is valuable. The growth should be transplanted or excised.

The old operation of ligation, which is barbarous and unscientific, should not be performed. The surgical management of this disease will be discussed in the latter part of the present chapter. In extensive pterygia, which cover a large part of the cornea, it may be advisable not to disturb the growth, but to perform iridectomy. Fortunately these neglected cases are seldom encountered. Removal of a pterygium is followed by increased haziness of the cornea, due to inflammatory action, and often this cloud disappears slowly. The best visual result does not always come immediately. Ophthalmometric examinations show that the corneal curvature is altered by the pterygium, and after its removal search should be made for astigmatism. Pterygium causes astigmatism in two ways: by lifting up the superficial corneal layers and by traction. Patients from whom pterygia have been removed often complain that the growths have returned. In a measure, this is true, but the secondary growth is smaller, and consists chiefly of blood-vessels which are necessary to the reparative process in the cornea.

Pseudopterygium (Cicatricial Pterygium) is the name applied to the fixation of a fold of conjunctiva upon the cornea as a sequence of inflammatory action. This may result from trauma by heat or caustics, or may follow operations for the removal of conjunctival growths. In some instances pseudopterygia develop after chronic superficial ulceration of the margin of the cornea. In other cases they are sequent to a chronic blennorrheic or to a diphtheritic process. A true pterygium ordinarily permits the passage of a sound beneath its folds at the limbus, while in pseudopterygium this cannot be done. In the former the growth generally is progressive, while pseudopterygia remain fixed at the part of the cornea to which they originally became attached. Small pseudopterygia may be permitted to remain; large ones should be excised. There is a rare form of false pterygium named pterygo-symblepharon, which is characterized by adhesion between the lower lid and the cornea through an intervening flat, vascular band of conjunctiva.

Pinguecula is a small, yellowish growth of the bulbar conjunctiva, adjacent to that part of the cornea corresponding to the interpalpebral fissure. It looks like a piece of fat, but is considered a hyalin degeneration of the conjunctiva and subconjunctival tissue, associated with an hypertrophy of the elastic fibres of the conjunctiva. It is rounded or triangular in shape, and is found in adults who are exposed to irritating influences. Fuchs considers that pterygium originates from pinguecula, but Knapp holds that this statement is too sweeping. Pinguecula is generally non-progressive and rarely calls for treatment. It can be excised or destroyed by means of the cautery.

Amyloid Degeneration of the Conjunctiva.—This rare disease, which was first described by Oettingen, of Dorpat, has been observed principally in Russia. The conjunctiva becomes hypertrophied, often projects between the lids, and is yellowish, wax-like, non-vascular, and friable. The disease

generally begins in the retrotarsal folds and invades the bulbar and palpebral portions of the conjunctiva as well as the caruncle. The eyelids are much thickened, and, when drawn forcibly apart, the wax-like conjunctiva is seen surrounding the cornea, which is usually clear, but may show pannus. The swollen tissue is friable, and often breaks when the lids are held apart. The disease often lasts for years without inflammatory symptoms, and the patient cannot see because of inability to open the lids. There is no discharge or lacrimation, and pain is absent. The affection is as frequently unilateral as bilateral. The cases so far reported have occurred in adults. Some writers have claimed that a relationship exists between trachoma and amyloid degeneration; but this view is not now accepted. The disease has nothing to do with amyloid degeneration located in other organs or tissues.

The first step is an hypertrophy of the conjunctiva. This is followed by an increase of the adenoid elements, and later there is a hyalin degeneration, which is followed by the appearance of amyloid bodies. Finally, calcification and ossification occur. Hyalin and amyloid degeneration look so much alike that differentiation can be obtained only by examining excised pieces of tissue. Calcification or ossification of the diseased conjunctiva may occur in these cases. In a doubtful case the diagnosis can be made by means of the iodin test. Treatment is of little value. Excision may be of benefit.

Xerosis (Xerophthalmos; Atrophy of the Conjunctiva).—In this condition the conjunctiva becomes thick, dry, and of a whitish color resembling skin. An analogous process is observed in the cornea. Xerosis of the conjunctiva appears either sequent to a local disease or as an accompaniment of a general affection. Locally it occurs after cicatricial degeneration following trachoma, pemphigus, burns, or diphtheritic conjunctivitis *(parenchymatous xerosis);* or it may be due to insufficient protection, as in ectropion and lagophthalmos, the exposed conjunctiva becoming covered with a thick, epidermoid epithelium *(epithelial xerosis).* Resulting from a general disease, conjunctival xerosis occurs in a light and also in a severe type. The former accompanies nyctalopia, while the latter is found in cases of keratomalacia. A special bacillus (xerosis bacillus) has been described. According to Fuchs, it is neither the cause of xerosis nor characteristic of this disease, since it is found in the healthy conjunctival sac.

In the primary type the part of the conjunctiva corresponding to the palpebral opening shows a froth-like deposit (composed of degenerated epithelium), which is triangular in form, the base being toward the cornea. The conjunctiva is anesthetic and irritation fails to produce lacrimation. In severe cases the cornea becomes dull and opaque and may slough. An important subjective symptom is night-blindness. The disease is common in Brazil and among the negroes of the South. Prognosis is unfavorable.

TREATMENT.—Primary xerosis of the conjunctiva is found chiefly among the ill nourished and calls for supportive and tonic treatment.

Local treatment is of little value. Glycerin and water, an emulsion of codliver-oil, or white vaselin may be used as local applications with comfort to the patient.

Tuberculosis of the Conjunctiva.—Since Koster, in 1874, described this disease (Fig. 8, Plate VIII) many cases have been recorded; but the bacillus of tubercle has been recognized as the cause only since 1882. It is a rare affection, Eyre having met with it in 8 out of 25,000 new ophthalmic cases in London. It may be primary, but is generally secondary to nasal or laryngeal tuberculosis. An abrasion from a slight trauma or the breaking of a conjunctival phlyctenula furnishes a *nidus* for the growth of the bacillus which can be carried to the raw surface by particles of dust, towels, fingers, or by unclean instruments used in tenotomy operations. The youngest case occurred in an infant of ten months, the oldest in a man of thirty years. Most cases occur at or about puberty. Females seem to be more liable than males in the proportion of 1.5 to 1. The palpebral conjunctiva is more often the seat of the lesion than is the bulbar portion. The lower lid is more often involved than the upper. The disease is generally unilateral. The cornea often is the seat of superficial inflammation. Iritis may develop. While the pre-auricular gland on the affected side is hard, swollen, and tender, it does not often suppurate. The submaxillary and cervical glands may be infiltrated.

The clinical features of the disease have been grouped by Sattler as follows: First group—characterized by small miliary ulcers, which may coalesce, generally attacking the palpebral, but sometimes affecting the bulbar conjunctiva. Second group—characterized by grayish or yellowish subconjunctival nodules, varying in size, but rarely larger than a hempseed. Third group—characterized by florid, hypertrophied papillæ and rounded outgrowths of granulation tissue, springing from the palpebral conjunctiva or situated in the fornices, recurring after removal, and accompanied by edema and thickening of the lids. Fourth group—"lupus" of the conjunctiva, characterized by numerous pedunculated, cockscomb-like excrescences in the fornices, of a jelly-like consistency, often showing more or less ulceration. To these Eyre adds another group to cover those cases characterized by distinctly pedunculated tumors, microscopically resembling papillomata: cases without involvement of the subconjunctival tissue or production of any symptoms other than mechanical ones. Pain, as a rule, is not a prominent symptom. A moderate discharge is present.

Etiology.—This disease is due to the tubercle bacillus, as can be shown by microscopic sections and by inoculation experiments.

Diagnosis.—Tuberculosis of the conjunctiva may be mistaken for trachoma, papilloma, chancre, or epithelioma. Trachoma cases improve under treatment by silver, while tuberculosis does not. In case of doubt a microscopic examination of an excised portion of infiltrated tissue should be made. In the absence of the tubercle bacillus inoculation experiments should be undertaken. This method is more reliable than microscopic

examination, although it requires a longer period (six or eight weeks) before the diagnosis can be determined.

PROGNOSIS.—This will depend on the ability of the surgeon to remove all of the diseased tissue. Where this is not practicable a cure should not be expected, although vision may be lost by involvement of the cornea. Spontaneous cure of conjunctival tuberculosis is possible.

TREATMENT.—Stephenson has cured one case by x-ray treatment. If this means fails, early and complete removal of the diseased conjunctival tissue and enucleation of swollen pre-auricular glands should be undertaken. This can be done with the knife, spoon, or cautery. Any tuberculous material which is left behind may cause general infection. After the diseased tissue has been removed a collyrium of bichlorid of mercury, or a dusting-powder, such as iodoform or aristol, can be applied. The general system should receive proper treatment. Good food, fresh air,

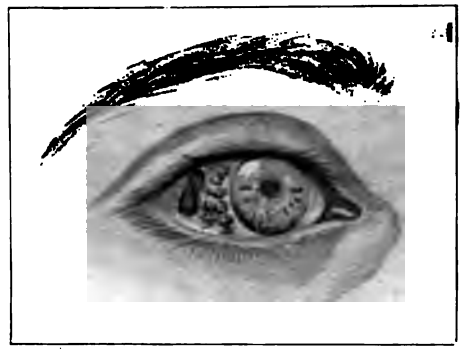


Fig. 214.—Tuberculosis of the conjunctiva. (EYRE.)

Granulations are present on the temporal side of the bulbar conjunctiva of the right eye, with a central caseous mass.

and proper climatic conditions should be secured. Tuberculin injections have not proved efficient in this disease.

Argyrosis of the Conjunctiva.—As a result of the long-continued application of a solution of nitrate of silver or of protargol, or from exposure to the action of silver-dust, the conjunctiva becomes discolored. Prolonged use of sulphate of iron will cause a yellow coloration of the conjunctiva (siderosis conjunctivæ). An example of argyrosis is shown in Fig. 7, Plate XI. Silver is deposited in the elastic fibres of the conjunctiva in the form of an albuminate or an oxid. The discoloration of silver causes the conjunctiva to appear bluish, while the stain following protargol is of a dirty-brownish color. There is no remedy for this condition.

Leprosy. — According to Neve, conjunctivitis occurs frequently in lepers, the anesthetic lids being exposed to the bites of flies and mosquitoes and to injury from foreign bodies. Leprous nodules occur in the conjunctiva by extension from the deeper tissues. They generally develop near

the corneal margin and grow into the sclera and cornea. The process extends and causes iritis, nodules appearing in the iris.

Lupus of the Conjunctiva may appear primarily, but is generally due to extension from the skin of the eyelid. In the conjunctiva it appears as an ulcer which may heal in one direction and extend in another. The lupous ulcer presents a bottom covered with granulations in which the bacillus tuberculosis has been found. Since the same microörganism is present in both tuberculosis and lupus, the diagnosis must be made by the clinical appearances. The treatment consists in excision, destruction by the cautery, or the application of the x-rays.

Hemorrhage from the Conjunctiva in rare instances becomes alarming, and, in the case of infants, has caused death. It may occur in persons who give no history of injury or of hemophilia. It has been seen to follow the operation of expression in trachoma, and also occurs without known cause. In at least three instances fatal hemorrhage has followed the application of nitrate of silver to the eyes of the newborn (Wiener). Profuse and repeated hemorrhages may occur for several months and suddenly cease. They are probably due to the presence of minute vascular tumors. When caused by an ulcer the bleeding vessel can be easily controlled by the application of forceps. If produced by minute vascular tumors the source of the hemorrhage may not be easily found. The affection is generally due to a lesion in the lower lid.

Abscess of the Conjunctiva, a localized area of suppuration in the subconjunctival tissue, may be due to trauma, but sometimes occurs apart from injury or other determinable cause. It may be situated at any part of the bulbar conjunctiva, but is more likely to involve the region of the caruncle. A suppurating Meibomian cyst opening through the palpebral conjunctiva is not uncommon. Pus from an orbital abscess may cause the conjunctiva to bulge and form a fluctuating mass. These conditions, however, are not abscesses of the conjunctiva in the strict sense of the term. Hot applications and an early incision is the treatment to be employed.

Actinomycosis of the Conjunctiva is a very rare condition which has been observed by Demicheri and Vincentiis. In the case reported by the former observer, a young man, suffering with subacute catarrh of the conjunctiva, presented on the everted upper lid, along the posterior tarsal margin, about fifteen yellowish-gray granules, three-tenths of a millimetre or more in diameter, resembling a granular or follicular eruption or the infarcts of Meibomian glands common in elderly persons. Microscopic examination of the contents of these granules showed typical masses of actinomycosis. The focus of infection was excised, with recovery.

Syphilis of the Conjunctiva.—The conjunctiva is subject to the primary, secondary, and tertiary lesions of syphilis: chancre, macular and papular syphilides, copper-colored spots, mucous patches, gummata, and ulcer. Chancres not only involve the conjunctiva by extension from the eyelids, but have also been seen primarily in the upper or lower fornix

or on the bulbar conjunctiva. Chancre (Fig. 7, Plate VII) here gives rise to the same characteristic symptoms as elsewhere. There is a small swelling and induration, ulcerated at the top, absence of pain, and enlargement of the lymphatic glands. The chancre feels like a piece of parchment. Buckley found over 4 per cent. of extragenital chancres located on the lids and conjunctiva. Infection occurs chiefly by the unclean finger of the patient or by the diseased mouth of another person. The latter source may be immediate, as in the removal of foreign bodies under the conjunctiva by the tongue; or intermediate, as by soiled towels, gloves, the wearing of a mask, the use of an opera-glass as in Falcone's case, or the use of unclean surgical instruments. The diagnosis of chancre of the conjunctiva is not difficult provided the surgeon thinks of the possibility of its occurrence, but the condition may be mistaken for gummatous ulcer, epithelioma, tuberculosis, or chalazion. The prognosis is favorable, only a small scar remaining. The local treatment includes cleanliness and the use of a mild mercurial ointment.

Mucous patches, which occur rarely on the conjunctiva, look like the same lesion elsewhere. Under internal and local treatment they generally heal rapidly. Copper-colored spots are rarely seen, and grouped papular syphilides are also of rare occurrence and are found in connection with the same lesions of the face and eyelids. Gummata of the conjunctiva are rare, and, according to de Beck, usually are developed in the ridge where the conjunctiva passes into the cornea, but may occur in other parts of the mucous membrane. They form rounded tumors of the size of a split pea. They are smooth and firm, present a light-pink color, and when uncomplicated cause no pain. They grow rapidly, and quickly disappear under proper general and local treatment. In the absence of a specific history they may be mistaken for cyst, abscess, or chalazion. These cases should be treated with iodid of potassium internally and cleansing washes locally.

Conjunctival Ulcers, aside from those occurring as a part of a tubercular or malignant process, are probably of more frequent occurrence than has been generally supposed. These lesions, which are often situated about one millimetre from the cornea and measure from two to four millimetres in diameter, may appear in any part of the membrane, but the bulbar conjunctiva corresponding to the interpalpebral space is a favorite site. The disease may cause marked injection, slight swelling, and little pain. Such a lesion will be overlooked easily unless a solution of fluorescin is used to outline it. Where only the epithelium has been cast off the stain does not show as well as in deeper ulcers.

The chief causes of conjunctival ulcers are trauma and syphilis. Some cases follow the breaking down of abscesses due to measles, variola, and other exanthematous diseases. Others are the result of local necrosis, such as occurs in diphtheria of the conjunctiva. The condition may be mistaken for acute catarrhal conjunctivitis.

Subconjunctival Ecchymosis (Subconjunctival Hemorrhage) is a common condition which follows injuries or operations or may occur spontaneously. It consists of a collection of blood beneath the ocular or transitional part of the conjunctiva. It can be distinguished from inflammation by its uniformity in color and by the absence of vessels. Subconjunctival hemorrhage is frequent after fractures of the skull. It is common in elderly people whose arteries are atheromatous, and often occurs in children with pertussis. The condition does not call for any particular treatment. The blood will be absorbed in from ten to twelve days.

Emphysema of the Conjunctiva occurs from the same causes that produce a similar condition of the lids, viz.: fracture of the nasal bones or of the walls of the frontal or ethmoidal cells, or ulceration of the bones leading to perforation of these spaces. It may result from forcible blowing of the nose. The swelling caused by emphysema is tense, elastic, and crepitates on pressure. It may accompany ecchymosis or may exist alone. The proper treatment is the application of a compress bandage.

Chemosis is a condition in which the ocular portion of the conjunctiva becomes edematous and is lifted up around the cornea, the swelling being often so great that the membrane projects between the eyelids. Such swellings may be classified as active, or inflammatory, and passive, or non-inflammatory. The former is present in almost all cases of purulent conjunctivitis. It follows the bites of insects and the stings of bees. It is occasionally seen in acute catarrhal conjunctivitis. It is of common occurrence in acute glaucoma, iritis, iridocyclitis, and in infection following operations on the globe. It is also present as a symptom of nephritis, and is sometimes caused by the internal use of certain drugs, such as iodid of potassium and quinin. The passive form of chemosis is seen in old alcoholics and in gouty persons.

A form of edema known as filtration chemosis occurs when the aqueous humor escapes beneath the conjunctiva through a fistula at the corneo-scleral margin.

The pathologic condition in chemosis is simply an infiltration of the subconjunctival tissue with leucocytes, transuded blood, and fibrin. There is also a formation of new vessels. The condition disappears with the subsidence of the cause, and does not call for treatment except when the swelling threatens the integrity of the cornea. In this event the swollen tissue may be incised in numerous places with a cataract-knife. The small cuts are to be made radial to the cornea.

Symblepharon, an abnormal adhesion of the eyelid to the globe (Fig. 4, Plate VII), results from the union of two raw surfaces, and may be caused by trauma, such as cuts or burns, or may result from pemphigus or diphtheritic or purulent conjunctivitis. It is rarely seen as a congenital condition. The attachment is generally between the lower lid and the globe. It may consist of a few slender threads of thickened conjunctiva or may exist as a dense band of tissue. Anterior symblepharon is an adhesion forming a bridge

between the lid and globe, but not reaching to the fornix; in the posterior variety the band involves the fornix. As a result of symblepharon there is limitation of ocular movements, and thus diplopia may be produced. When the adhesions involve the centre of the cornea vision will be reduced. Symblepharonic eyes are often irritated and hyperemic from constant traction. In some cases the lids are fastened to the globe in such a way that closure is impossible, and thus lagophthalmos is caused, with resulting corneal mischief. Often there is also entropion and trichiasis. Mild cases of symblepharon give rise to few or no symptoms.

The term symblepharon has been applied also to the contraction and shrinking of the conjunctiva following trachoma. Here there is no adhesion of opposing surfaces, but a slow diminution in size of the *cul-de-sac.*

The prognosis of symblepharon depends on the extent to which adhesions have taken place. In anterior symblepharon the prognosis is favorable; in the posterior form it is grave, and in total symblepharon little improvement is to be expected. When caused by shrinking, the condition is incurable. The treatment is surgical. Various operations for its relief are described in the latter part of this chapter.

INJURIES TO THE CONJUNCTIVA.

It is necessary to consider the lodgment of foreign bodies and the effect of trauma and of chemicals.

Foreign Bodies often lodge upon the conjunctiva, and are of the most varied nature. They cause pain, photophobia, lacrimation, and blepharospasm. When in the lower *cul-de-sac* they are easily observed. If in the upper fornix they are often overlooked, and can be seen best when secondary eversion of the lid is performed or when the lid is lifted away from the globe while the patient looks downward. The foreign body may rest on the conjunctiva or become fastened in the membrane, either by the force which originally propelled it or by the action of the patient in rubbing his eye. A foreign body may be washed away by the tears. If found loose in the conjunctival sac, it can be removed by flushing the eye with warm physiologic salt solution. If attached, it can often be removed by wiping the conjunctiva with a toothpick wrapped in absorbent cotton. The foreign body becomes caught in the cotton, and thus is not lost. When deeply lodged in the upper fornix foreign substances may remain *in situ* for many months, producing much or little discomfort, and leading to a luxuriant growth of granulations. Insects, bugs, and larvæ may lodge in the *cul-de-sac* and often cause intense irritation, owing to the action of the formic acid which most of them contain. In all cases where the presence of a foreign body is suspected, the search for it should not be abandoned until after the upper fornix has been explored.

Burns of the Conjunctiva (Fig. 1, Plate X) are frequently caused by lime, pieces of hot metal, or gunpowder. Scalds often follow the bursting of

water-gauges on locomotives. The gravity of such injuries is often unappreciated at the time of the receipt of the injury. Among the results are ankyloblepharon, symblepharon, entropion, ectropion, as well as lesions of the cornea and destruction of the lids. The raw conjunctival surfaces grow together and lead to extensive adhesions. In the treatment an attempt should be made to prevent the union of raw surfaces. If the lower *cul-de-sac* has been deeply burned, adhesion will be sure to occur and the fornix will be obliterated. Burns of the upper *cul-de-sac* without destruction of the eyelid are rarely seen.

The treatment of burns of the conjunctiva should be begun at the earliest possible moment. Acids can be neutralized by a 1-per-cent. strength solution of sodium bicarbonate or potassium bicarbonate (saleratus). Hot metal should be removed as soon as possible. In such injuries the injection of castor-oil into the *cul-de-sac* is often advised. In burns by lime the offending substance should be removed as early as possible, and to accomplish this a jet of cold water is to be employed. The use of water in such cases has long been denounced by ophthalmic writers, but recently Andreae has shown that cold water neutralizes the heat generated by the contact of lime with tears, and aids in the speedy removal of the lime. Contrary to popular belief, water in contact with oxid of calcium does not generate sufficient heat to injure the eye within ten minutes. It may happen that the pieces of lime can be removed more rapidly by the use of a spatula, knife-blade, or spud. After removal has been accomplished holocain can be used to relieve pain and iced compresses can be applied to control inflammatory action. The adhesion of raw surfaces must be prevented by passing a probe into the fornices twice daily. Vaselin can be used within the *cul-de-sac.* If these measures are insufficient, Coover and Black advise the use of egg-film placed between the raw surfaces. The film, taken from the small end of an egg is to be placed over the globe and changed daily. In case the cornea is involved the use of atropin will be in order.

In injuries by gunpowder the eye should be anesthetized by holocain or cocain and the powder-grains should be picked out by means of a cataract-needle or snipped off with scissors. In the after-treatment the daily use of a solution of boric acid will be in order. In such cases the cornea often is involved and atropin must be used. Often the powder-grains will be driven through the cornea and into the iris or lens.

Trauma of the conjunctiva, aside from the lodgment of foreign bodies or burns, must be considered. The membrane may be torn by blows from the fist, the rent being often situated in the bulbar conjunctiva concentric with the cornea. Such cases require cleanliness, a suture, and cool applications.

AFFECTIONS OF THE CARUNCLE.

The Plica Semilunaris and Caruncle participate in inflammations of the conjunctiva. These parts are frequently red and swollen in persons

suffering from eyestrain, and particularly in those with imperfect convergence. The older writers gave considerable attention to a localized inflammation of the caruncle which they named *encanthis*. It is attended by infection and swelling of the sebaceous glands, and may lead to suppuration, in which case the small abscess should be opened.

Excessive development of hairs on the caruncle, *trichosis carunculæ*, may cause persistent hyperemia, with the sensation of a foreign body. Epilation or excision of the hair-bulb is the proper treatment.

Tumors of the Caruncle. — As primary growths, adenomata, papillomata, cylindromata, fibromata, lymphangiomata, sarcomata, and carcinomata have been seen in this region. They should be excised. Congenital telangiectasiæ and dermoid cysts have also been observed. Lithiasis of the caruncle is a rare affection. The concretions should be picked out with a cataract-needle.

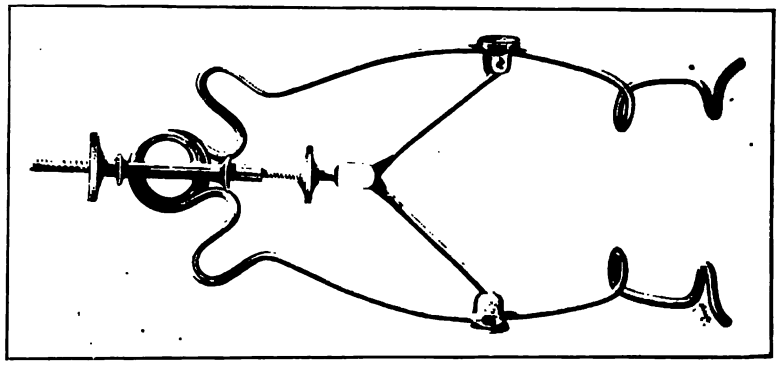


Fig. 215.—Eye-speculum. (AUTHOR.)

OPERATIONS ON THE CONJUNCTIVA.

The chief operations performed on the conjunctiva are those for pterygium, symblepharon, trachoma, and tumors. Such minor procedures as the removal of a foreign body from or the opening of an abscess of the conjunctiva, or the application of medicines to this membrane need no further mention in this place. In all operative procedures limited to or involving the conjunctiva it will be necessary to follow certain rules regarding the preparation of the field of operation. The necessity is none the less patent although the impossibility of rendering the conjunctiva sterile is now recognized. The skin of the face, forehead, and eyelids must be washed with hot water and soap, then with bichlorid solution (1 to 5000). In case pus or muco-pus is present in the lacrimal sac the secretion must be removed and the passages flushed with the same solution. The conjunctiva is to be cleansed by causing warm sterile water to flow over its entire surface, the upper lid being everted. This is followed by a flushing with bichlorid (1 to 5000). The eye is then to be covered with a sterile towel wrung out of the same solution. The conjunctiva is then anesthetized with

sterile holocain or cocain solution, the medicine being conveyed by a sterile dropper.

Pterygium Operations.—A pterygium may be treated by (1) excision, (2) transplantation, or (3) cauterization. The instruments needed are a stop-speculum, fixation forceps, dissecting forceps, sharp-pointed scissors, strabismus-hook, needles, needle-holder, sutures, a cataract-knife, and an electrocautery.

1. The Excision Operation is begun by seizing the pterygium near the corneoscleral margin and lifting the growth up. The apex can then be shaved off from the cornea. If the surgeon prefers divulsion a small cut is made beneath the neck of the growth with sharp-pointed scissors and a strabismus-hook is passed beneath. The divulsion is accomplished by tearing in the direction of the cornea. It is claimed that this procedure gives a better separation between the pterygium and the cornea than can be obtained otherwise. The apex being freed, the body of the growth can be separated from the normal conjunctiva back as far as the caruncle, and then excised. The gap is filled by uniting the adjacent conjunctiva with interrupted sutures. This operation is applicable only to small pterygia. In large, fleshy ones, particularly in those with expanded bases, total excision is not advisable, since the motion of the eye in the opposite direction may be limited by the cicatrix. In such cases it will be best to excise only the apex and a part of the body of the growth or to resort to transplantation. Complete excision of the pterygium, the denuded area being covered with a Thiersch flap, has been successfully practiced by Hotz.

2. Transplantation is done by first separating the apex as described above. The growth is then to be split in the direction of the long axis, one half being transplanted into the upper, the other into the lower, fornix. The raw space made by the separation of the growth from the eyeball is filled by suturing the adjacent conjunctiva. To facilitate sliding of the membrane, incisions are to be made concentric with the cornea. A gauze dressing is to be applied and the sutures are removed on the fourth day. There are several modifications of the transplantation operation, that of McReynolds being preferred by the author. The apex of the growth is lifted up from the cornea and is separated from it by shaving it off with a von Graefe knife. Then the pterygium is to be thoroughly lifted from the underlying sclera for a distance of five millimetres from the cornea. An incision is made along the lower border of the growth for a distance of ten to twelve millimetres. The ocular conjunctiva is separated from the globe below the growth, the separation extending to the lower fornix. The apex of the pterygium is to be flattened and unrolled with forceps, while two needles are passed through the apex of the growth and are brought out deeply in the fornix, as is shown in Fig. 216. On tying the suture the pterygium is buried and at the same time the denuded scleral area is covered with conjunctiva. The suture is removed at the end of a week.

3. Cauterization.—This (Coe's) method, according to Loring, is a

valuable procedure. It consists simply in the application of the cautery to the apex of the pterygium on the cornea, the body of the growth not being touched. The operation is said not to be followed by pain or inflammatory symptoms. One thorough application is said to be sufficient.

Symblepharon Operations.—Procedures for the relief of symblepharon anterius are successful, while operations for symblepharon posterius frequently are failures. In the former condition the adhesion is severed close to the eyeball, and reunion is prevented by daily use of the probe. In case

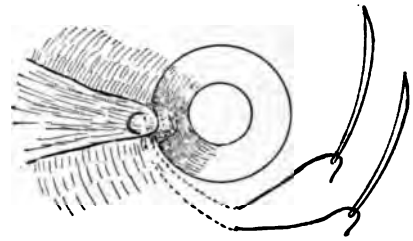


Fig. 216.—Operation for pterygium. (McREYNOLDS.)

the symblepharon extends from the cornea back to the fornix, the ingenuity of the surgeon will be taxed. Among the best operations for this condition are the procedures of Knapp, Arlt, Teale, and Harlan.

In Knapp's method, after adhesions have been severed, the defect is covered with vertical, stretched flaps of conjunctiva, which are stitched into the fornix. In Arlt's operation the corneal part of the symblepharon is detached and is stitched into the fornix by threads passed through the lid, while the denuded surface is covered by sliding the conjunctiva from each

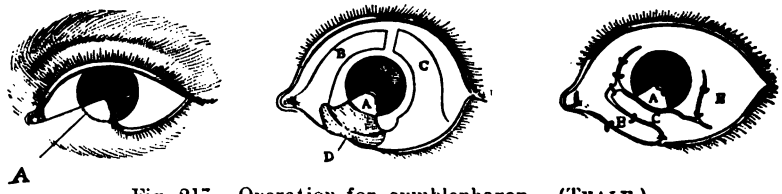


Fig. 217.—Operation for symblepharon. (TEALE.)

A, Tip of the symblepharon. *B*, *C*, Conjunctival flaps. *D*, Denuded surface left by removal of the symblepharon from the eyeball.

side. This method of disposing of symblepharon insures a conjunctival surface opposed to the raw spaces if the conjunctiva cannot be stretched sufficiently to cover the defect. If such a contingency arises it will be best to cover the raw surface with a bit of mucous membrane from the patient's mouth, or a piece of rabbit's conjunctiva, or a skin-graft after the manner of Thiersch.

In Teale's operation the tip of the symblepharon is left *in situ* and the remainder is dissected from the eyeball down to the fornix. The defect is closed by conjunctival flaps, as shown in Fig. 217.

Harlan's operation (Fig. 218), which the author has used successfully in total symblepharon of the lower lid, is performed in this manner: The eyelid is separated from the globe and a bridge is made of the lid by an incision *(A-B)* parallel to its margin. An incision *(C-D)* serves to loosen a skin-flap, which is turned inside out, drawn beneath the bridge, and stitched by its raw surface to the raw surface of the lid. The space left by the turning of the flap can be closed by sutures, an incision *(C-E)* being made to relieve tension on the tissues.

Skin-grafting in Symblepharon.—Total symblepharon of one eyelid, a condition formerly supposed to be incurable, has recently furnished a more favorable prognosis, cases having been operated upon successfully by May, Hotz, and others. Formerly mechanical devices were used to prevent reunion of the cicatricial bands, but of late surgeons have learned that no permanent good result can be secured unless the raw surfaces are covered with epithelium. Mechanical devices (shields of glass, porcelain, lead, and silver) are now used to hold large Thiersch grafts in position until they have become united to the underlying tissue.

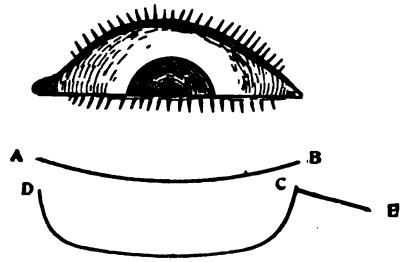


Fig. 218.—Operation for symblepharon. (HARLAN.)

Removal of the Tarsus and Retrotarsal Folds.—This (Heisrath's) operation dates from 1882, and in recent years has come into use in the treatment of trachoma, owing to Kuhnt's enthusiastic advocacy of it. The operation is begun by everting the upper lid, which is seized by two fixation forceps and drawn strongly upward. This exposes the junction of the ocular and palpebral conjunctiva, along which line a curved horizontal incision is made through the conjunctiva only, from canthus to canthus. Three stitches are now passed through the bulbar conjunctiva; the fixation forceps is now released, and the lid is held everted over a spatula while an incision is made parallel with and from three to five millimetres from the ciliary border. The conjunctiva and tarsal plate are then dissected carefully from the orbicularis muscle. The wound is irrigated with bichlorid solution and closed by the sutures already passed through the bulbar conjunctiva. To obtain a satisfactory result the sutures must be passed through corresponding points in the wound-margins.

Excision of the Upper Cul-de-sac, an operation for the cure of trachoma which was reintroduced by Galezowski in 1874, did not become popular

until after the publications of Brachet (1882) and Despagnet (1884). Hotz, Jaesche, and Lloret condemned the operation. Lately it has come into use largely by reason of the opinion of Stephenson, who performed excision of the upper fornix on more than seventy eyes, and saw only one untoward result, his cases having been under observation for periods varying from two to six years. After a successful excision a transverse line of scar-tissue marks the site of operation, the fornix being represented by a shallow depression. The ocular movements are not limited and ptosis is not usually induced. The upper fornix is removed, because it is the seat of the tissue in which the trachomatous process is most marked and least amenable to topical treatment. Removal of this tissue lessens the danger of recurrence of the trachomatous process and of pannus.

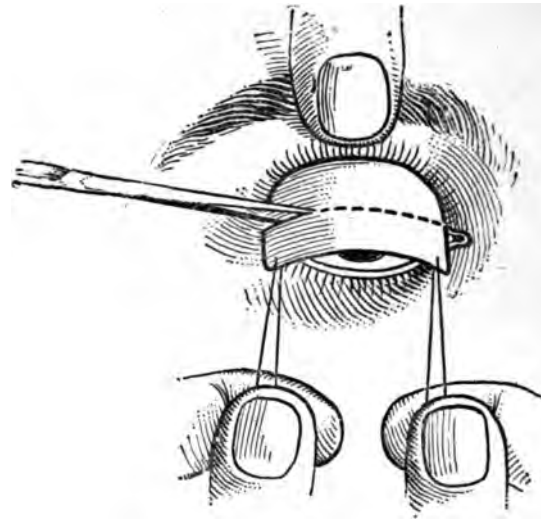


Fig. 219.—Excision of the upper cul-de-sac. (After STEPHENSON.)

The operation is done under local anesthesia. The upper lid being everted, the fornix is seized and drawn downward. At each extremity of it a needle is passed bearing a silk suture, whose ends are held taut by an assistant. With scissors the conjunctiva is separated from its attachment to the tarsal plate. The subconjunctival tissue is then dissected and the operation is completed by cutting through the posterior layer of the *cul-de-sac*. Free bleeding during the operation may necessitate torsion of a few vessels. Sutures are not used to close the wound. The reaction is usually slight. If large granulations appear in the wound during healing, they can be cut off with scissors. Ptosis may follow the operation and soon disappear.

Expression of the Contents of Trachoma Follicles, an old method of treatment, has been popularized by the articles of Hotz, Noyes, and Knapp. The procedure requires two pairs of roller forceps, the instrument of Knapp

being one of the best. Under cocain or general anesthesia the upper lid is everted and seized with the forceps, one pair being used to steady the lid while the other is employed to roll out the trachomatous material. Care should be taken to reach all parts of the fornices. After the squeezing process is finished cold applications are used for two or three days, followed by appropriate local treatment.

Grattage of the Conjunctiva.—This operation, which many French ophthalmologists recommend for trachoma, requires a pair of fixation forceps, a three-bladed scarifier, a tooth-brush, and a solution (1 to 500) of bichlorid of mercury. The everted lid being held by forceps, the surgeon thoroughly scarifies the conjunctiva and scrubs the incised surface with a stiff brush soaked in the bichlorid solution. Cold applications are to be used for a few days. A probe is used daily to prevent the formation of adhesions between the palpebral and ocular parts of the conjunctiva.

Periectomy.—As the first step in this operation, which is done for pannus, a piece of bulbar conjunctiva two or three millimetres wide is removed close to the peripheral border of the pannus. Then a corresponding strip of subconjunctival tissue is excised. The underlying sclerotic, which Boeckmann claims is always the seat of scleritis in these cases, is scarified with a cataract-knife until it appears of a normal whitish color and the vascularization of the cornea disappears. This step is usually followed by a copious arterial hemorrhage. The last step is to arrest this hemorrhage and provide for an open, broad wound. If the conjunctiva is permitted to cover the scleral wound, the object of the operation will be defeated, the aim being to form a scar which shall be a bar between the corneal and pericorneal inflammation. To prevent the conjunctiva becoming attached, the eye is turned in the opposite direction and the wound is filled with powdered iodoform, while hemorrhage is controlled by pressure with sterile gauze, the pressure being continued for half an hour. The operation is followed by increased vascularization of the cornea; but this gradually disappears, and in three or four weeks the condition of the cornea is much improved.

Peritomy (Syndectomy; Tonsure of the Cornea).—A less formidable and also less efficient operation than periectomy is peritomy. Under local anesthesia a strip of conjunctiva is removed from around the limbus. Blunt scissors are used to cut the membrane. A strip three to four millimetres wide is removed adjacent to the cornea. A small Volkmann scoop is then used to remove the subconjunctival tissue. A gauze dressing is applied, and healing is uneventful. This operation was formerly much in vogue in the treatment of pannus.

Subconjunctival Injections.—After cleansing the conjunctival sac a local anesthetic is used and the lids are held apart by the fingers of an assistant or preferably separated by a speculum. Then with forceps the conjunctiva is lifted up at a point six to eight millimetres from the limbus and the needle of a hypodermic or Pravaz syringe, filled with the fluid, is

passed to the depth of two or three millimetres in a direction parallel with the sclera. The fluid is then discharged, 2 to 10 minims being injected. The process may be repeated at intervals of two or three days.

INDICATIONS.—Subconjunctival injections are valuable in the treatment of inflammations of the iris and ciliary body, in sloughing ulcers of the cornea, in scleritis and episcleritis, and in detachment of the retina. They are of little, if any, value in chronic keratitis, in chorioiditis, in retinitis, and in optic neuritis. The subconjunctival injection of cocain is used in patients requiring enucleation or evisceration who cannot or will not take a general anesthetic.

SOLUTIONS USED.—Bichlorid and cyanuret of mercury, trichlorid of iodin, sodium chlorid, and hetol (cinnamate of soda) are the substances which are most frequently used at the present day. Many others have been tried and abandoned. As regards strength, bichlorid is used from 1-30,000 to 1-1000; trichlorid of iodin, 1-2000 to 1-500; sodium chlorid, 0.75-1000 to 2, 5, 10, or 20 per cent.; hetol, 1 per cent.

ACCIDENTS.—In spite of the use of a local anesthetic, severe or excruciating pain not infrequently follows subconjunctival injections. According to Darier, the addition of a few drops of a 1-per-cent. strength solution of acoin to the medicament to be used subconjunctivally will render the injection painless. Subconjunctival ecchymosis may follow the injury to a vessel. Injury to the sclera will not occur if reasonable care is used. Localized necrosis of the conjunctiva may occur after a subconjunctival injection of a bichlorid solution.

CHAPTER VIII.

DISEASES OF THE CORNEA.

SINCE the cornea is the most exposed part of the eyeball, it is particularly liable to injury. Devoid of blood-vessels, except at the extreme periphery, it readily becomes necrotic under the influence of pathologic processes. Hence it is not surprising to find that corneal diseases form a large percentage (25 to 33) of ophthalmic affections, and that of 10,000 blind persons Uhthoff found that over 13 per cent. had lost their sight by corneal diseases. If to these are added the cases in which purulent conjunctivitis produced corneal complications, the percentage of blindness from corneal disease is more than 27. Hence the great importance of corneal injuries and diseases to the patient, to the general practitioner of medicine, and to the ophthalmic surgeon. Although the layers of Bowman and Descemet possess great resisting power, all parts of the cornea are susceptible to pathologic influences. Unfortunately for ophthalmic writers, the making of a rational division of corneal diseases is as yet impossible, since there is no way of approaching these affections according to a fixed basis: *i.e.*, one that is etiologic or anatomic.

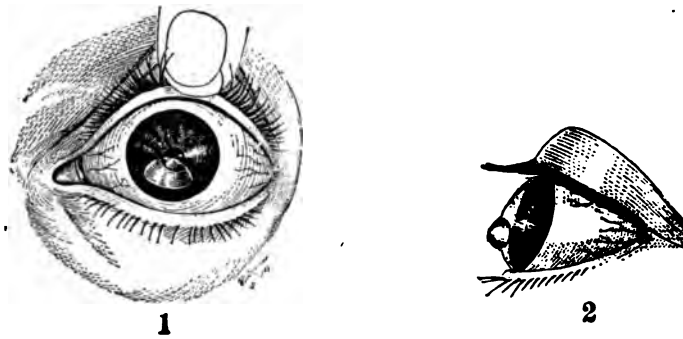
CONGENITAL ANOMALIES.

Opacities often occur in connection with anomalies of size and form of the cornea. In some instances corneal clouds are the only demonstrable changes. Such opacities may be stationary or progressive after birth, or may disappear entirely. Whether these opacities are due to an arrest of development or to an intra-uterine inflammation is a question which is still under dispute, although most of the late authors incline to the latter view. The form of the opacity presents many variations: sometimes there is a circular cloud involving the whole of the limbus (embryotoxon); or several sickle-shaped opacities are found; or tongue-shaped opacities occupy the periphery of the cornea, resembling the residuum of the so-called sclerosing keratitis of extra-uterine life; or a diffuse, grayish-white, dense opacity, looking like porcelain, involves either the whole cornea or the central portion. In many of the cases last mentioned alterations in shape (keratoconus or keratoglobus) are present; or there is an increase of intra-ocular tension leading to hydrophthalmos. Such dense opacities arise from the posterior surface of the cornea. If only a simple cloud is present, the lesion is in the endothelium. In enlargement and ectasia of the cornea the existence of an ulcer of the posterior surface is to be assumed (von Hippel).

Pigmentation.—Kayser saw a patient with congenital green coloration of the periphery of the cornea.

Congenital Ectasia is a rare malformation which Pincus attributes to inflammation occurring in the second half of fetal life. It is probably due to an arrest of development in the fetal cleft, forming a coloboma of the cornea, which in the later weeks of intra-uterine life becomes filled with clear corneal tissue. In Würdemann's case (Fig. 220) a small "segment was implanted on the cornea like a little oval watch-crystal, five millimetres high and seven millimetres in width, perfectly clear and of higher refraction, so that the subjacent portion of the iris appeared magnified." This condition has been misnamed congenital staphyloma. The term staphyloma is applied only to those cases in which involvement of the iris coexists with protrusion of the cornea.

Hydrophthalmos will receive consideration in the chapter on "Glaucoma."



1 2

Fig. 220.—Congenital ectasia of the cornea. (WÜRDEMANN.)

1, Front view. *2*, Lateral view.

Megalocornea and Keratoglobus are terms used synonymously to indicate enlargement of the cornea. The difference between them and hydrophthalmos should be clearly understood. In megalocornea the eye shows no signs of internal disorganization. There is no excavation of the optic nerve, and good vision may be present. In hydrophthalmos there is excavation of the nerve-head, internal disorganization of the eye, and loss of vision.

Megalophthalmos is a rare congenital condition in which the eye is enlarged in all its diameters and pathologic changes are absent.

The congenital conditions here mentioned, with the possible exception of hydrophthalmos, do not admit of treatment.

TUMORS OF THE CORNEA.

Primary tumors of the cornea are extremely rare, most of the growths involving this structure having their origin in the conjunctiva. Among the primary corneal growths which have been reported by competent observers are papillomata, fibromata, myxomata, sarcomata, epitheliomata, dermoids, and keloids. Snellen, Galezowski, Stellwag, Colsmann, Lagrange,

and Treacher Collins have met with epitheliomata of the cornea. Rumschewitz, Panas, and Pagenstecher have reported cases of primary sarcoma of the cornea. Silex saw a fibroma. The author has observed one case of symmetrically placed tumors of the corneæ, which histologic examination showed to be fibromata. Westcott met with a keloid of the cornea occurring in a babe with staphyloma. Similar cases have been reported by R. Simon and I. Szokalski. Corneal cysts also occur. They are usually implantation cysts and follow perforating injuries. Collins met with one situated partly in the cornea, partly in the sclera, which measured 9.5 by 5 millimetres and was lined with laminated epithelium. Generally corneal cysts are too small to be seen macroscopically, and are found on microscopic examination of enucleated eyes. Schieck has observed the formation of four superficial corneal cysts in a case of blennorrhea with great chemosis. He believes that a superficial marginal keratitis was followed by the formation of pseudopterygia, beneath which the cysts developed.

Treatment.—Since they principally occur in eyes demanding enucleation, the treatment of corneal tumors is practically *nil.* If the growth is observed in an early stage, it should be removed and subjected to microscopic examination. The prognosis will depend upon the laboratory findings.

INFLAMMATION OF THE CORNEA.

Inflammation of the cornea (keratitis, corneitis) may be acute or chronic, idiopathic or traumatic, congenital or acquired, primary or secondary. It may or may not end in ulceration. Keratitis may be simply a local process leading to abscess and ulcer; or the local manifestation of a constitutional disease, as the parenchymatous keratitis of inherited syphilis; or it may be only a part of a disease—such as kerato-iritis and sclerokeratitis—involving deeper portions of the eye. In keratitis the cardinal signs of inflammation—heat, swelling, redness, pain, and loss of function—are all absent early in the disease. The existence of keratitis is then evidenced only by cloudiness. This lack of transparency is due to infiltration by leucocytes which have passed into the cornea from the adjacent bloodvessels or have originated from the fixed corneal cells by karyokinesis. Cloudiness of the cornea impairs vision, and often it is this which causes the patient to consult the surgeon. In cases of keratitis which go on to ulceration, pain, photophobia, circumcorneal redness, and hyperemia of the conjunctival vessels are marked symptoms. The existence of a corneal infiltrate is shown by the presence of cloudiness and a lack of lustre at the affected spot, but the surface is not changed in curvature: *i.e.*, it is not uneven. The infiltrate, in favorable cases, disappears by resorption; in unfavorable ones it ends in suppuration, in which case suppuration may be limited to the internal layers *(corneal abscess)* or may destroy the corneal tissue external to the abscess and thus leave a *corneal ulcer.* This ulcer is described as "foul" when its walls are still infiltrated, or "clean" when the

surrounding cloudiness has disappeared and its base is transparent. An ulcer which is covered by epithelium reflects light: *i.e.*, is clear and bright. The chief clinical sign of a corneal ulcer is unevenness of the corneal surface, which shows an excavation. Corneal suppuration means a loss of substance, and this loss is repaired by a new-tissue formation which is not corneal, but connective tissue, and hence is opaque. Hence, a corneal opacity means a corneal scar.

The source of pus in suppurative keratitis was for many years a subject for controversy. It was formerly erroneously believed that corneal corpuscles could proliferate to form pus-cells. While "the corneal corpuscles about an ulcer may proliferate to some extent before becoming necrotic, the new cells thus produced are but few as compared with the immigrated leucocytes" (Holden).

In its normal state the cornea is devoid of vessels except at its extreme periphery, but under pathologic conditions vessels are rapidly developed, being necessary to the reparative process. After this is complete the vessels diminish in size and number and may entirely disappear. It is important to know the situation of vessels in the cornea, since this furnishes an index of the kind of keratitis present. *Superficial vessels* are clearly seen. They spring from the marginal loops of vessels in the limbus, and can be traced from the cornea to the limbus and thence to the conjunctiva. They branch arborescently. Pannus may be taken as a type of superficial vascularization. Here the vessels, for the most part, do not lie in the cornea, but in a newly formed tissue resting on the cornea. *Deeply seated vessels* are not clearly recognizable, since they are clouded by infiltrate in the superficial corneal layers resting over them. They are of a reddish-gray color and arise from the vessels of the sclera close to the margin of the cornea. They seem to end suddenly at the corneal margin; and in branching they form ramifications running parallel with one another, like the straws of a broom. Parenchymatous keratitis shows this type of vascularization.

The cornea having to sustain the intra-ocular tension, it follows that any pathologic process which softens this membrane renders it liable to the danger of alteration in its curvature. Thus, keratitis is often followed by *staphyloma*. Neighboring tissues participate in the pathologic process in keratitis; thus, there is conjunctival and ciliary hyperemia. Iritis and iridocyclitis are common in severe cases, and pus forms in the anterior chamber *(hypopyon)*. While a small hypopyon may undergo resorption, a large one is likely to cause corneal necrosis and loss of vision.

The origin of hypopyon has been the subject of much controversy. Ewing states that it has been attributed to the direct passage of leucocytes through Descemet's membrane (Horner, Bokowa); to the traveling of leucocytes around Descemet's membrane and their passage into the anterior chamber via the ligamentum pectinatum (Schweigger); to leucocytes originating in the endothelium (Hoffman); to direct rupture of Descemet's

membrane (Weber, Verdese, Silvestri); to leucocytes from the canal of Schlemm and small, deep, circumcorneal vessels (Nuel); and to exudation from the iris (Arlt). Uhthoff and Axenfeld, after a series of careful investigations, doubt the origin of hypopyon from the cornea, and attribute it to the iris and ciliary body. Their conclusions have been generally accepted by modern ophthalmologists; but old pathologic ideas die hard, and some surgeons still believe that hypopyon originates from a direct break in Descemet's membrane and in an exudate from the iris. Elschnig has attributed the breaks in Descemet's membrane to erosions produced by chemotactic changes in aggregations of leucocytes on the posterior surface of the cornea, the leucocytes being derived from the iris, ciliary body, and ligamentum pectinatum. Two points in favor of this view are the absence of bacteria in hypopyon and its frequent occurrence with intact membrane of Descemet.

Onyx, a term much used by the older writers to describe the shape of an hypopyon, is now applied to a collection of leucocytes in the corneal stroma or beneath Descemet's membrane. It occurs chiefly in the lower part of the cornea. Frequently hypopyon and onyx are both simultaneously present in the same eye. Onyx does not move when the patient changes his position, while hypopyon, if liquid, does change its position. Yet it often occurs that hypopyon is thick, gelatinous, and immovable. In some cases the differentiation between these conditions can be made by oblique illumination.

CLASSIFICATION OF CORNEAL INFLAMMATIONS.

Although no absolutely correct classification of corneal inflammations is possible in the present state of science, for teaching purposes it is advisable to set before the reader a subdivision of these diseases. Since a classification founded on etiologic factors is impossible, these inflammations may be divided according to their location (as superficial and deep), or according as the process of infiltration undergoes resorption or advances to suppuration (non-suppurative and suppurative keratitis). For practical purposes the latter method is preferred, and the subjoined classification is submitted with the understanding that it is open to criticism:—

(A) Suppurative Keratitis.

1. Ulcer of the cornea.
2. Creeping ulcer of the cornea.
3. Neuroparalytic keratitis.
4. Lagophthalmic keratitis.
5. Xerophthalmic keratomalacia.

(B) Non-suppurative Keratitis.

1. Phlyctenular or eczematous keratitis.
2. Interstitial keratitis.
3. Vascular keratitis.
4. Bullous and vesicular keratitis.
5. Aspergillar keratitis.
6. Malarial keratitis.
7. Filamentary keratitis.
8. Keratitis punctata.
9. Superficial punctate keratitis.
10. Ribbon-shaped corneal opacity.
11. Herpes of the cornea.
12. Marginal keratitis.
13. Striped keratitis.
14. Disc-like keratitis.
15. Grill-like keratitis.

(A) SUPPURATIVE KERATITIS.

Ulcer of the Cornea.—When a corneal infiltrate does not undergo resorption it ends in suppurative keratitis. It must be understood, however, that non-suppurative and suppurative inflammations merge into one another by insensible degrees. If the suppuration leads to a circumscribed collection of pus between the corneal lamellæ, the condition is known as *corneal abscess*. If, in this condition, the superficial corneal layers undergo necrosis, so that the abscess communicates with the external world, the case is one of *corneal ulcer*. In the life of an ulcer several stages are recognized. When the ulcer is spreading and its edges are "foul," we speak of the state of progression; when it begins to become "clean" and shows signs of healing, the stage is that of regression; and, when it undergoes healing, we speak of the stage of cicatrization. This last stage invariably leaves a scar of greater or less extent, and this interferes with the transparency of the cornea. If the process of ulceration is unchecked, it leads to perforation of the cornea, with such important sequelæ as iris-prolapse, the formation of anterior synechiæ, or anterior polar cataract, and in some instances destruction of the eye by panophthalmitis. Microörganisms are necessary to corneal suppuration, and these are generally introduced from without, finding lodgment in some minute spot of corneal tissue from which the epithelium has been exfoliated or removed by trauma. The warmth and moisture found about the eye are factors favorable to the growth of bacteria. Corneal wounds which are promptly treated and dressed aseptically rarely lead to suppuration. Unfortunately there are no means of rendering the eye absolutely aseptic, compatible with the integrity of its tissues.

Perforation occurring in the course of an ulceration of the cornea is a result of several factors, such as increased intra-ocular pressure from sneezing or crying, from blepharospasm, or from increased blood-pressure. A sharp pain and a sudden escape of aqueous humor are the subjective signs of perforation. Objectively it is indicated by minus tension of the eye, obliteration of the anterior chamber, with or without the presence of iris-prolapse. Previous to the occurrence of perforation, when the ulcer has acquired great depth, there may be a projection of the membrane of Descemet, in the form of a transparent vesicle *(keratocele)*, which fills the floor of the ulcer and may advance beyond the level of the cornea. If the perforation is small and central, the iris may not become prolapsed, but the lens is pushed forward and closes the opening. An exudate is poured out, and, after the aqueous humor reaccumulates and the lens resumes its normal position, a tag of exudate is found adhering to the anterior capsule of the lens. Thus there is formed an *anterior polar cataract*. In such cases repeated ruptures of the cicatrix may be followed by a permanent *fistula of the cornea*. While a perforation may be looked upon as an alarming accident, as a matter of fact its influence upon corneal ulceration is generally favorable, by reason of the sudden reduction of tension permitting an improvement in the circulation of corneal fluids.

PLATE XI.

External Diseases of the Eye.

Fig. 1.
Interstitial Keratitis.

Fig. 2.
Creeping Ulcer of the Cornea
(Ulcus Serpens).

Fig. 3.
Blood-staining of the Cornea.

Fig. 4.
Staphyloma of the Cornea.

Fig. 5.
Dendritic Keratitis.

Fig. 6.
Panophthalmitis.

Fig. 7.
Large Corneal Scar. Iridectomy.
Argyrosis of the Conjunctiva.

Fig. 8.
Phthisis Bulbi.

PLATE 11

In case of perforation located outside the centre of the cornea, the iris becomes attached at the site of perforation, and remains there, forming an *anterior synechia.* The plugging of the perforation permits the rapid restoration of the anterior chamber, and, under favorable circumstances, healing takes place with the formation of a flat cicatrix. Under unfavorable circumstances, the prolapsed iris and cicatricial tissue are too weak to resist intra-ocular pressure, and a bulging scar, *staphyloma of the cornea,* results. Where practically the whole cornea perforates, there is a *total prolapse of iris.* Among the accidents following perforation are luxation or extrusion of the lens, the occurrence of intra-ocular hemorrhage, purulent irido-cyclitis, and panophthalmitis. In cases where these disastrous complications do not ensue, and where healing progresses favorably, a corneal scar results, which may be more or less opaque for many months or years.

ETIOLOGY.—Corneal ulcers are classified etiologically as primary and secondary. The former begin in the cornea, while the latter are due to conditions located originally in tissues outside of the cornea. Primary ulcers result frequently from trauma of slight degree. The trauma may be the lodgment of a small foreign body which is driven into the cornea by the force of wind or by an explosion. It may come from the constant friction of misplaced cilia or by the rubbing of the roughened conjunctiva upon the globe. It may be due to wounds received accidentally or by operations; or, finally, it may be caused by burns, scalds, or escharotics. Secondary ulcers result from inflammations of the conjunctiva, disturbances in the trigeminal nerve, or the existence of grave typhoid states. Extreme protrusion of the eyes in exophthalmic goitre may lead to inability of the lids to protect the corneæ, which slough.

As regards age, corneal ulcers are exceedingly rare in children, except as a feature of phlyctenular keratitis or as following purulent, diphtheritic, variolar, or croupous conjunctivitis. In middle-aged and elderly persons they are very common. It is probable that such individuals possess slight resisting power to the invasion of bacteria, which are ever present in the conjunctival *cul-de-sac,* and are important factors in the etiology of corneal diseases. The microörganisms may be the ordinary pus-cocci, the gonococcus of Neisser, the pneumococcus, or the colon bacillus. Serpiginous ulcers with hypopyon are almost invariably due to pneumococcic infection. Ulcers not typically serpiginous are generally due to the staphylococcus and streptococcus, but may result from the pneumococcus. In rare instances the sloughing ulcer is caused by schizomycetal infection: *Aspergillus fumigatus.* As regards social position, ulcers of the cornea are much more common among the poor and laboring classes than among the rich, the middle, and the professional classes. This is due to two factors: deficient nutrition and the liability to trauma among these unfortunates.

VARIETIES OF ULCERS OF THE CORNEA.—Ulcers of the cornea present many clinical variations. Aside from the catarrhal ulcers which are mentioned on page 253, and phlyctenular ones which will be considered under

the head of "Phlyctenular Keratitis," there are others, viz.: the simple ulcer, the round central ulcer, the rodent ulcer, the crescentic ulcer, the dendriform ulcer, the atheromatous ulcer, and the serpent ulcer.

The Simple Ulcer, which may be secondary to the rupture of a phlyctenula, or primarily caused by trauma, presents itself as a small, superficial opacity, with loss of corneal substance and slight pericorneal injection. It may be simple or multiple, and tends to recur. While generally healing without incident, in the ill nourished it may be converted into a deep purulent ulcer. Under the use of a boric acid solution these ulcers often heal readily. If due to phlyctenular disease, the ointment of the yellow oxid of mercury should be employed. In any event, attention to the general health will be in order.

The Round Central Ulcer, sometimes called the central, non-irritative ulcer, is indolent and stationary, often remaining for months. It is round and is located over the pupil. Often it is clear at the bottom, but may show some infiltration. It is characterized by the absence of irritative symptoms. Vessels are commonly absent, and photophobia and lacrimation are not marked symptoms. Such ulcers occur in cases of chronic catarrhal conjunctivitis and in trachoma. They may remain superficial or may become deep and end in perforation or in the formation of a permanent scar. As regards treatment, any conjunctival disease present must receive appropriate attention. This, combined with the daily use of a 1-per-cent. strength solution of boric acid, and the protection of the eyes by dark glasses, will suffice. Atropin is generally unnecessary in this form of ulcer.

The Rodent Ulcer (ulcus rodens of Mooren) is an extremely rare condition, and occurs chiefly in advanced life. It generally develops at the corneal margin as a narrow, extended, superficial ulcer with noticeable inflammatory symptoms. It has undermined edges. The surrounding cornea is infiltrated, and from the limbus numerous vessels pass to the diseased area. Hypopyon is rare in rodent ulcer and perforation does not occur. Other infiltrated spots appear and undergo ulceration. These involve about one-third of the thickness of the cornea. They do not perforate, and are separated from the normal tissue by a grayish undermined margin. In healing they leave a dense cicatrix. Soon other ulcers appear within the zone first attacked, and the process continues until the centre of the cornea, the last part to be involved, is affected. Commonly both eyes are involved and the patient is left blind. The course of the disease is slow and may last for months. As regards etiology, Andrade has examined two cases and has found a special bacillus which he believes is the pathogenic factor. The condition yields to the galvanocautery. Measures less heroic are useless in this disease.

The Ring Ulcer, which appears, for the most part, in aged and decrepit subjects, begins near the limbus and extends around the circumference of the cornea. Thus, the nutrient supply is cut off and the whole cornea sloughs. Pain is not a prominent symptom in these cases. A common

cause of ring ulcer is trauma. The disease is frequent among miners. A similar ulcer is sometimes seen in children as a result of phlyctenular disease.

The only remedy for ring ulcer is destruction of the infected area, preferably by the galvanocautery. Eserin in weak solution (gr. $^1/_4$ to ℥j) is useful. Hot applications can also be employed with benefit, but poultices are never in order. If perforation is threatened, eserin should be instilled and paracentesis should be performed. Later in the history of the case, or after the galvanocautery has been employed, atropin will be in order to check iritis. Tonics, antiperiodics, and, in suitable cases, stimulants are to be used. In the ring ulcer of children it will be necessary to use an ointment of the yellow oxid of mercury locally and to give attention to the general health.

The Crescentic Ulcer, which generally appears at the upper part of the cornea, begins near the limbus. It is painful; photophobia and lacrimation are marked symptoms. If unchecked, it leads to perforation. Usually it is due to catarrhal conjunctivitis. In simple cases the treatment of the affected conjunctiva by solutions of silver or of protargol or of argyrol will be required.

The Dendritic Ulcer will be described under the name of "Malarial Keratitis."

A form of suppurative keratitis which, on account of its seriousness, demands special consideration, is the creeping, serpentine, or serpiginous ulcer.

The Atheromatous Ulcer is found in old corneal scars and in staphylomatous eyes which have undergone hyalin degeneration with deposition of lime salts. Small pieces of lime may be found lying on the floor of the ulcer under a thick coating of pus. These ulcers tend to perforation and the consequent production of panophthalmitis. The atheromatous ulcer is primarily a necrosis, thus differing from the ordinary ulcer, which is primarily an infiltration. It differs from ulcus serpens in this respect, that the necrotic process extends only so far as is necessary for the sequestration of a given portion of the corneal tissues (Fuchs).

Creeping Ulcer of the Cornea (Hypopyon-Keratitis—Roser; Necrotic Corneal Abscess—Weber; Ulcus Corneæ Serpens—Saemisch; Serpiginous Ulcer).—This form of ulcer begins as a rounded spot of a yellowish or grayish-white color occupying the central portion of the cornea (Fig. 2, Plate XI). Its tendency is to spread peripherally. The opacity is greater at the margin of the ulcer than at the centre. In the area of propagation the margin is elevated and more yellow than in other places. Surrounding the ulcer is a zone of gray infiltration presenting striæ. That part of the cornea corresponding to the ulcer is at first somewhat elevated, but soon becomes depressed. A violent iritis and the early appearance of hypopyon attest the severity of the pathologic process. Pain, photophobia, circumcorneal injection, and slight edema of the lids are present in most cases. If the process is unchecked, great sloughing occurs with corneal per-

foration, and vision is correspondingly reduced or entirely lost. When perforation occurs, the hypopyon and aqueous humor are evacuated and the iris prolapses. In favorable cases perforation is followed by a scar, which may be flat or ectatic; in unfavorable ones panophthalmitis ensues, and the eye ends in phthisis bulbi. In favorable cases a dense scar is left in the cornea, the iris is adherent to the lens, and the pupil is occluded by a membrane. In unfavorable cases the destructive process is rapid.

Etiology.—Creeping ulcer of the cornea may be found at any period of life, but is most frequent between the fortieth and seventieth years. Of 80 cases occurring in the Basel ophthalmologic clinic, reported by Wehrle, 22.5 per cent. were poorly nourished, while 77.5 per cent. were in good condition. In 56 cases traumatism was present; in 3 exposure to cold was the supposed cause, and in 21 cases the cause could not be determined. Many of the patients presented chronic conjunctival and lacrimal diseases. Creeping ulcer is comparatively frequent among miners, vine-trimmers, harvesters, and others who are exposed to accidental injuries. As regards its bacteriology, it will suffice to say that recent investigations by Druault,

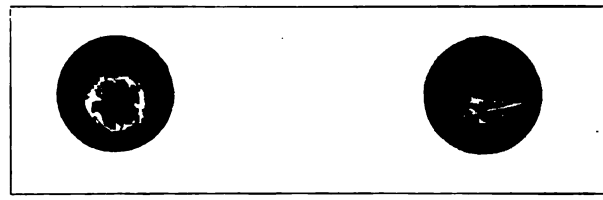


Fig. 221.—Appearance of the cornea in ulcus serpens. (KNIES.)

A, In the height of the disease. *B*, After treatment by Saemisch's incision.

Petit, Coppez, Morax, Axenfeld, and others show conclusively that most cases of serpiginous ulcer of the cornea, and all typical cases, are due to the pneumococcus, which is found between the layers of the membrane. Other bacteria, particularly the staphylococcus and streptococcus, cause ulcers which are clinically similar to the pneumococcic ulcer. As mentioned above, trauma can be traced in a majority of serpiginous ulcers; and it is safe to assume that, in the remainder of the cases, slight injuries to the epithelial layer have permitted the bacteria to begin their invasion. In all cases of ulcerous keratitis there is microbic infection which is exogenous in type. Whether endogenous microbic infection of the cornea is possible is not known.

In a large percentage of cases of ulcus serpens the patient suffers from trachoma or from a chronic inflammation of the lacrimal sac. In some instances he carries the infection by his handkerchief or by using his saliva to moisten the eye. Ulcus serpens also occurs in variola, rubeola, typhus, scarlatina, etc. Fuchs states that the ulcus serpens of variola appears not in the height of the disease, but in the stage of desiccation;

and, like the ulceration occurring in other infectious diseases, has been attributed to metastasis, but is more likely due to infection from without. Variolous ulcers are found in children as well as in adults, and often both eyes are affected.

Pathology.—This form of ulcer spreads both laterally and in depth. It often leads to perforation. Early in the history of the case intra-ocular disturbances are present, such as turbidity of the aqueous humor, the presence of a fibrinous exudation on the lens and iris, detachment of the endothelium behind the ulcer, and the presence of a purulent exudation in the anterior chamber. Leucocytes are abundant in the tissues about the circumcorneal zone, in the anterior chamber, the ligamentum pectinatum, the posterior lamellæ of the cornea, the endothelial layer, and in the iris.

Fig. 222.—Creeping ulcer of the cornea. (Author.)

(Photomicrograph by Dr. H. P. Wells.)

The extending ulcer presents overhanging margins. It may extend in one direction while healing in another. Numerous foci of inflammation appear, scattered over its floor. The corneal lamellæ slough, down to the layer of Descemet. This rarely breaks down early. Generally it bulges forward as a transparent bleb, filling in the floor of the ulcer. When it ruptures, the aqueous humor escapes, the cornea collapses, and the lens either comes forward, blocking the rupture, or it escapes entirely from the globe. Perforation may cause intra-ocular infection, ending in panophthalmitis, or the morbid process may show signs of improvement and the eyeball be saved.

Prognosis of Corneal Ulcers.—However small and insignificant it may appear, a corneal ulcer is always an element of danger to visual acuity and even to the integrity of the globe. Early in the case the prognosis is

usually favorable. However, in cases of purulent conjunctivitis, in keratomalacia, and in serpiginous ulcers great damage will often result in spite of early and intelligent treatment. The prognosis of "foul" and extending ulcers is unfavorable, while, if the ulcer becomes "clean" the outlook is favorable. When, in the course of a corneal ulcer, vessels extend from the limbus into the floor of the ulcer, the prognosis is good.

DIAGNOSIS.—The presence of an ulcer of the cornea is determined by inspection by natural or artificial light, with or without the aid of a magnifying glass. In cases of blepharospasm the use of holocain will facilitate the examination. In children it often is necessary to use lid-retractors. To determine the exact limits of an ulcer the surgeon can use a 2-per-cent. strength solution of fluorescin, which stains the denuded part of the cornea a greenish tint, in marked contrast to the surrounding normal tissue.

TREATMENT OF CORNEAL ULCERS IN GENERAL.—In the treatment of corneal ulcers the principles to be kept in mind are: 1. Removal of the cause. 2. Limitation of the pathologic process and prevention of perforation. 3. Promotion of the reparative process. 4. Removal, so far as possible, of the effects of ulceration.

1. The removal of foreign bodies lodged in the cornea or conjunctiva, or of cilia rubbing on the globe, will be in order. Since many cases of corneal disease are dependent on affections of the conjunctiva, it will be more necessary to direct treatment to the latter than to the former.

2. To limit the pathologic process and prevent perforation is usually an easy task if the patient comes early; but under other circumstances it may be impossible. The chief agents in limiting the spread of an ulcer are various antiseptics and caustics, such as bichlorid of mercury, formalin, tincture of iodin, carbolic acid, and, best of all, the galvanocautery. The use of atropin solution is valuable because of its effect on the accommodation and blood-vessels, and because it also prevents iritic adhesions. On account of its constitutional effects it must be used cautiously in children. In cases where the pupil does not enlarge under its application, particularly where old and firm synechiæ are present, atropin will best be dispensed with, for the reason that it may produce increased intra-ocular tension. In deep peripheral ulcers which are likely to perforate it should not be used; in such cases eserin or arecolin will be better than atropin. As a rule, the use of a bandage is to be prohibited. It never should be employed where secretion is profuse, since here it becomes moistened and hot, and acts as a poultice. The chief arguments in favor of a protective bandage are that it keeps dust from the eye and prevents movement of the globe. In order to immobilize the affected eye, however, it will be necessary to bandage both eyes. Poultices, which are so popular with the laity, have no place in modern ophthalmology. They are not only useless, but often are positively harmful. The eyes should be protected from light by smoked glasses. The intermittent application of moist heat is often of benefit. It can be applied by pouring boiling water on a towel and placing this, when somewhat cooled,

upon the closed eyelids, leaving it in position for two to five minutes and repeating; or the same effect can be obtained by pouring the water into a glass brim full, and having the patient immerse his eye with the lids closed. These applications of moist heat can usually be repeated every three or four hours with benefit. They relieve pain and allay congestion. The collyria used should generally be soothing and non-astringent. Collyria leading to a metallic deposit in the cornea, such as lead acetate, should not be used.

Where perforation seems impending, daily paracentesis of the cornea, the application of a bandage, and the use of a miotic to reduce intra-ocular tension will be in order. The subconjunctival injection of a solution of bichlorid of mercury, which has been advocated by Reymond and Secondi, of Turin, and Darier, of Paris, is an efficient means of checking corneal necrosis and clearing up an hypopyon. After the local use of cocain, the injection is made, a Pravaz syringe being used. The solution may vary in strength. Gargarin, of St. Petersburg, used a $^1/_{10}$-per-cent. solution; Secondi uses a solution composed of 1 part of sublimate and 2 parts of sodium chlorid dissolved in 2000 parts of water. The amount to be injected varies from a few drops to one-half of the capacity of the Pravaz syringe. Darier injects a solution of cyanid of mercury (1 to 4000). To make the injection painless, he uses cocain or holocain locally and adds a few drops of a 1-per-cent. strength solution of acoin to the cyanid solution. These injections are sometimes followed by burning pain lasting for an hour, edema of the bulbar conjunctiva, and swelling of the upper eyelid. Necrosis of the conjunctiva at the site of the injections may ocur. On account of these untoward effects of sublimate injections, Mellinger and others have abandoned the sublimate for a normal salt solution, which is efficient and non-irritating. Wehrle, in comparing the effects of linear cauterization, subconjunctival injections of sublimate, and subconjunctival injections of salt solution in the treatment of hypopyon keratitis, gives preference to the salt solution. If, under the plan of treatment outlined above, the ulcer continues to spread, it will be advisable to use the galvano-cautery. Its application is often followed by marked improvement, a foul ulcer becoming clean and repair ensuing. If the surgeon does not wish to use the cautery, he can curette the ulcer and apply tincture of iodin, a strong solution of nitrate of silver, or a drop of pure carbolic acid. A 10-per-cent. solution of pure nitric acid, applied by means of a wooden applicator, is a valuable caustic for serpiginous ulcers. In this kind of an ulcer the operation known as Saemisch's incision is often followed by great improvement. In this procedure a cataract-knife is passed through the cornea and the hypopyon is evacuated either spontaneously, or, if thick, is seized with forceps and drawn out.

3. The reparative process is promoted by the measures just described. Moist heat favors the separation of necrotic from living tissue, promotes the necessary development of new blood-vessels in the cornea, and relieves pain. It is particularly valuable in subacute ulcers. Puncture of the base of the

ulcer or paracentesis external to it, in cases of impending perforation, is often useful, since it reduces tension, starts a flow of lymph into the cornea from its nutrient vessels, and promotes repair. The same effect results from the corneal incision of Saemisch in serpiginous ulcers. Chandler and Risley have found the use of a 10-per-cent. ointment of cassaripe, a vegetable antiseptic, of great value in the treatment of corneal ulcers. The observance of ordinary hygienic rules and the use of tonics, antiperiodics, and sometimes of stimulants will be in order in corneal ulcerations in the aged.

4. The removal of the effects of ulceration must take into consideration the immediate effects (complications) and those which are remote (sequelæ). The complication most to be dreaded is perforation with prolapse of the iris. If the patient is seen shortly after the occurrence, it will be advisable to use a general anesthetic, draw the iris forward through the opening, and excise it close to the cornea. The angles of the iris-coloboma are then to be stroked back into place by means of a spatula. Gamo Pinto follows this procedure by placing an excised piece of conjunctiva, twice as large as the original opening, between the lips of the corneal opening. A compressing bandage is then applied. This method will sometimes result in the formation of a flat scar without anterior synechia. The treatment of other complications and of the sequelæ of corneal ulcers will be discussed elsewhere in this chapter.

Neuroparalytic Keratitis (Neuropathic Keratitis). — This name is applied to the condition of the cornea which follows paralysis of the ophthalmic division of the fifth nerve.

ETIOLOGY.—Paralysis of the ophthalmic division of the fifth nerve may be due to any one of many causes: tumors in the pituitary region or at the base of the brain; syphilitic, traumatic, or epidemic meningitis; caries of the temporal bone; fracture of the skull; primary neuritis, a rare cause; disease of the nuclei of the trigeminal nerve; or operation for removal of the Gasserian ganglion for the cure of neuralgia. Whether the lesion is located in the nerve-trunk or in its nuclei of origin in the brain, the result is the same. In paralysis of the fifth nerve winking and lacrimation do not occur; hence the cornea becomes dry and minute foreign bodies settle upon it. Infection takes place and destruction of tissue follows. Since the time of Magendie it has been customary to attribute neuroparalytic keratitis to the loss of the influence of trophic nerve-fibres supposed to be located in the trigeminus, but in the light of modern pathology it is no longer necessary to adopt this hypothesis. Snellen's explanation, that the insensibility of the eye occurring in paralysis of the fifth nerve enables ordinary causes to act unmolested on the cornea, is now generally accepted.

SYMPTOMS.—The chief symptom is anesthesia. In paralysis of the entire fifth nerve or of its ophthalmic branch, both cornea and conjunctiva become anesthetic. After optico-ciliary neurectomy the cornea alone is anesthetic, winking and lacrimation remaining and no harm resulting to the

cornea. If, however, the branch of the seventh nerve supplying the orbicularis muscle is paralyzed, the cornea is likely to slough. Neuralgic pain, through the region supplied by the affected ophthalmic branch of the fifth nerve, may precede the anesthesia. There may be loss of taste on one side of the tongue; and the motor root may also be involved, as is evidenced by paresis of the masseter, temporal, and external pterygoid muscles. Localization of the pathologic process is often possible by a study of the extent of the anesthesia and of the associated conditions. For this information the reader is to consult the standard works on neurology.

Following paralysis of the fifth nerve the cornea becomes cloudy, the epithelium of its centre is loosened and removed, and this process extends until only a narrow peripheral rim remains. The central ulcer is at first gray; then it becomes yellowish, hypopyon forms, the ulcer perforates, and the iris is engaged in the cicatrix. Healing is followed by a flat scar. Not every case runs a course so severe as this, since the process may not lead to perforation. In neuroparalytic keratitis ciliary injection is present, but lacrimation is absent.

The corneal changes following excision of the Gasserian ganglion can be prevented by stitching the lids together and at the first dressing applying a Buller shield. If these precautions are not observed, the cornea may necrose and the eye be lost. In necrosis and suppuration of neuroparalytic keratitis, pain, lacrimation, and blepharospasm are absent. Ocular tension is usually reduced.

Prognosis.—Neuroparalytic keratitis is always a serious disease. If treated early and properly the process can usually be checked and useful vision can be saved. In such cases, after restoration of tissue has occurred, an iridectomy may improve vision.

Treatment.—The preventive treatment has been mentioned above. The curative treatment consists in the application of a bandage, the use of atropin or eserin, and the frequent cleansing of the eye with an antiseptic solution. Electricity may be tried. Nieden advises the hypodermic injection of strychnin in the temple.

Lagophthalmic Keratitis (Keratitis e Lagophthalmo).—Keratitis from drying of the cornea due to imperfect closure of the eyelids may be attributed to many causes. It is found in paralysis of the orbicularis muscle in persons suffering with profound exhaustion (typhoid fever, uremic coma, etc.); in cases of destruction or cicatricial contraction of the eyelids; in caries of the orbital margin, producing ectropion; and rarely in exophthalmic goitre when the exophthalmos is excessive. As a result of desiccation the cornea becomes fissured, and pathogenic germs, gaining a foothold, soon cause suppurative inflammation. Hypopyon, iritis, and extensive ulceration of the cornea are common, while perforation and total loss of the eye are not rare. "The corneal ulcer is distinguished by its location at the lower edge of the cornea, by its sharply divided upper edge parallel to the margin of the upper lid, and, finally, by the fact that a dry scale is

first produced, the ulcer not being visible until the scale is thrown o (Fick). The general course of the disease resembles that of neuroparaly keratitis. The prognosis is serious, and particularly is it grave in t corneal ulceration of exophthalmic goitre.

TREATMENT.—If possible, the cause should be removed. The cor must be protected, and, in case a bandage does not suffice, the operat of uniting the eyelids (tarsorrhaphy) will be in order. It is customary make a partial union of the eyelids, the outer portion being usually selec for operative intervention, although it may be deemed advisable to ur the middle third of the lids.

While the operation of partial tarsorrhaphy has generally been regar as harmless, in cases of exophthalmic goitre it has sometimes been follo

Fig. 223.—Keratitis e lagophthalmo. (AUTHOR.)

(Original drawing by DR. CARL FISCH.)

by disastrous results. Jessup says: "I should hesitate to again employ in such cases, or at all events if a patient over thirty-five years old. soon as the cornea was affected in my case, no treatment seemed to have slightest effect in staying the ulceration." In milder forms of lagophthalr keratitis the affected eye should be bandaged at night. Atropin and m antiseptic washes are to be used and all sources of irritation are to avoided.

Xerophthalmic Keratomalacia (Xerotic Keratitis; Simple Kera malacia; Infantile Ulceration of the Cornea with Xerosis of the Conju tiva).—This rare disease, which was first described by von Graefe, usua appears in marasmic children during the first four years of life. In a c observed by the author, a boy, aged 3 years, after a prolonged attack

diarrhœa presented conjunctival xerosis of both eyes. Later the cornea became infiltrated near its periphery and also in the centre. The bulbar conjunctiva, particularly that part corresponding to the palpebral opening, presented a frothy, grayish, silvery appearance due to exfoliation of epithelium. There was diminished sensitiveness of the cornea and of the conjunctiva.

A form of xerotic keratitis occurring frequently among negro children has been described by Kollock, of Charleston, S. C. In his cases, which occurred chiefly among scrofulous, emaciated subjects, the interpalpebral part of the conjunctiva presented a dirty-white, yellowish-green hue. In some cases the ocular conjunctiva was thickened and discolored; in others it was relaxed and flabby and was thrown into folds about the cornea by the movements of the globe, and "occasionally these folds were capped by the silvery scales which some writers have described as being diagnostic of hemeralopia." In the disease under consideration night-blindness is rarely the cause of the child being brought to the surgeon, since the affection occurs in persons so young that usually they either do not go about unattended or cannot describe their symptoms accurately.

The chief danger in this disease is corneal ulceration. In some patients the corneal infiltration, which is present in all cases, does not go on to ulceration. When ulceration occurs it often assumes the form of a ring, which may be elevated or depressed. The centre of the cornea may or may not be clear. The whole cornea presents a bluish, hazy sheen, due to opacity of the epithelium, which in rare instances is raised into folds. Perforation of the cornea may occur. Iritis without perforation is rarely observed. Pain, photophobia, and lacrimation are absent in xerotic keratitis. Spicer has observed cases in which xerosis of the conjunctiva was absent.

Etiology.—The cause of this disease is unknown. It follows exhausting diseases, such as typhoid fever, meningitis, and chronic diarrhea. Various bacilli and cocci have been found in these cases, but it is not known whether they are etiologic factors. The disease is more common in Austria than in America. In this country it seems to occur more frequently among negro than among white children. Koller states that the disease is sometimes epidemic, especially in orphan asylums, and attributes it to reflection from a body of water or from a white wall.

Prognosis.—According to Swanzy and others who have observed the disease in European clinics, xerotic keratitis is almost always followed by death from exhaustion. In Kollock's cases benefit followed treatment and no mention is made of a fatal result.

Treatment.—The weakened constitution must be strengthened by tonics, stimulants, and proper food. Change of air and surroundings may be beneficial. As the strength grows the ocular condition will improve. Locally vaselin or a weak ointment of the yellow oxid of mercury (gr. ij to ʒiij), or a solution of boric acid may be applied. When there is corneal ulceration, atropin will be in order.

(B) NON-SUPPURATIVE KERATITIS.

Phlyctenular Keratitis (Eczematous, Scrofulous, or Strumous Keratitis; Fascicular Keratitis).—This disease, which should be studied in connection with phlyctenular conjunctivitis, is a common one, characterized by the development of phlyctenulæ on the cornea instead of the conjunctiva or limbus, as in phlyctenular conjunctivitis. Often both conjunctiva and cornea are involved at the same time. The causes of phlyctenular keratitis are the same as those of phlyctenular conjunctivitis. The disease is much more frequent in children than in adults, and is closely connected with disorders of the nasal passages (adenoid vegetations, hypertrophic or atrophic rhinitis, etc.). It often follows measles and other exanthemata.

Symptoms.—The first change in this disease is the appearance of collections of round cells beneath the epithelium, which is lifted up. This constitutes a corneal phlyctenula. It is usually situated at or near the corneoscleral junction. It varies from a poppyseed to a milletseed in size, and changes in color from gray to yellow in the course of a few hours. Soon it bursts, leaving a small ulcer,—*phlyctenular ulcer,*—and coincidently pain, photophobia, blepharospasm, and lacrimation become pronounced symptoms. The conjunctival and pericorneal vessels in the vicinity of the ulcer become engorged, and frequently there is a muco-purulent discharge from the conjunctiva. The ulcer may heal rapidly or may remain, other phlyctenulæ appearing and going on to the formation of ulcers. On the other hand, the process may disappear by absorption of the contents of the phlyctenula. If the ulcers heal before the deeper layers of the cornea are involved, only a faint nebula remains, and this in young subjects disappears entirely in the course of a few months or years. Often, however, the process is not favorable. The ulcers spread in breadth and depth, are surrounded by a deeply placed zone of grayish or yellowish infiltration, and may even lead to perforation of the cornea. Meanwhile vessels extend from the limbus to the ulcer, and these, when in the form of a leash or band, make part of a clinical picture which has been named *fascicular keratitis* (Fig. 8, Plate X). If a number of phlyctenulæ develop along the rim of the cornea, the condition may be named *marginal phlyctenular keratitis.* Instead of appearing as one or more clearly defined ulcers, the disease may present a thick tongue of vascularized tissue growing from the limbus toward the centre of the cornea—*pannus eczematosus.* Unlike the pannus of trachoma, this affection does not show a predilection for the upper part of the cornea, but often appears below or at the inner or outer side. Pannus eczematosus usually clears up satisfactorily under treatment, leaving only a slight opacity. The *single pustule,* which usually appears at the corneal periphery, is particularly dangerous, and is liable to cause perforation when surrounded by a yellowish, infiltrated area. Iritis and hypopyon often accompany it.

The intensity of the subjective symptoms is not a criterion of the gravity of the pathologic process. Often children present intense blepharo-

spasm, profuse lacrimation, and great photophobia, with relatively small corneal changes. The child keeps in the dark, buries the head in a pillow or the mother's lap, and screams when exposed to light. Under these circumstances inspection of the cornea is difficult and requires the use of lid-retractors. The reflex blepharospasm of this disease was explained many years ago by Iwanoff, who demonstrated the migration of round cells from the corneal margin to the pathologic focus under the epithelium. Thus, irritation of the nerve-filaments, whose course they follow (Fig. 224), is produced. A strange effect of long-continued blepharospasm, which has not been satisfactorily explained, is the occurrence of temporary amaurosis. In these cases there are no ophthalmoscopic changes. The patient usually regains vision in a few weeks. A persistent eczema of the nose, face, and ear, and chronic rhinitis, are common accompaniments of phlyctenular keratitis.

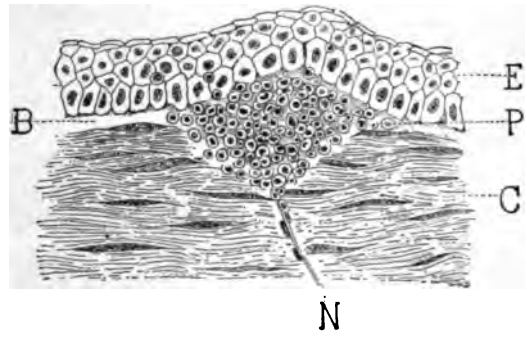


Fig. 224.—Section of the cornea in phlyctenular keratitis. (After IWANOFF.)

E, Epithelium. *P*, Round-cell infiltration. *C*, Substantia propria. *N*, Corneal nerve. *B*, Bowman's layer.

PATHOLOGY AND DIAGNOSIS are given under the heading of "Phlyctenular Conjunctivitis" (page 272).

PROGNOSIS.—If treatment is begun early the prognosis is favorable, healing occurring with only slight opacity. In severe types a perceptible scar remains. In neglected cases perforation of the cornea may occur.

TREATMENT.—The regulation of the diet and attention to the general health, as well as the treatment of naso-pharyngeal conditions will be in order here as in phlyctenular conjunctivitis (see page 274). Locally atropin drops and ointment of the yellow oxid of mercury (gr. j to 3j) should be used daily and should be continued for some time after all signs of the disease have disappeared. Darier claims that a 1-per-cent. strength solution of dionin, by its lymphagogic and analgesic properties, is of value in the treatment of phlyctenular keratitis.

Interstitial Keratitis (Specific, Parenchymatous, or Strumous Keratitis; Keratitis Profunda).—This is a chronic inflammation of the middle and posterior layers of the cornea, occurring chiefly in children, not leading

to ulceration, but accompanied by inflammation of the uveal tract. There is in these cases a deposit of new material in the cornea and a development of new blood-vessels.

Etiology.—From the records of many large clinics it is found that hereditary syphilis is the cause of interstitial keratitis in from 40 to 97 per cent. of the cases, thus confirming the opinion which was expressed by Hutchinson many years ago. The disease also may occur late in the history of acquired syphilis. Certain cases of interstitial keratitis are due to herpes zoster ophthalmicus. It is supposed that some cases of interstitial keratitis are of tuberculous origin (von Hippel). Although it may be an intra-uterine disease, interstitial keratitis generally occurs between the fifth and twen-

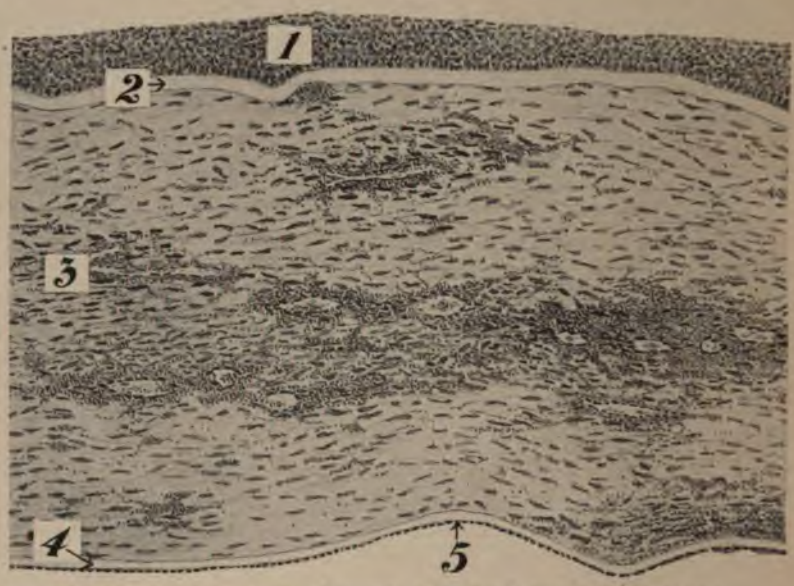


Fig. 225.—Section of the cornea in interstitial keratitis. (Baas.)

1, Epithelium. *2*, Bowman's layer. *3*, Area of infiltration. *4*, Membrane of Descemet. *5*, Endothelium.

tieth years. It is rarely observed after the thirtieth year. It is seen oftener in females than in males. In the victims of hereditary syphilis slight causes—such as eyestrain, irritants, rheumatism, and other acute diseases—suffice to produce the initial changes of interstitial keratitis.

Symptoms.—Patients with interstitial keratitis often show the signs of hereditary syphilis. Chief among these are the following:—

1. A peculiar physiognomy. "The conformation of the face is somewhat angular, the features are contracted and drawn, the skin is coarse, and the complexion is pale and earthy. The forehead is prominent, and the skin covering it is thrown into wrinkles through the frowning incidental to the long suffering from fear of light. The bridge of the nose is depressed, and may be completely sunken, through the loss of the bones from

syphilitic ulceration; from a similar cause affecting the skin, the angles of the mouth and the alæ nasi are scarred and fissured by white cicatrices." (Ramsay.)

2. The teeth are notched or pegged in 60 or 70 per cent. of the cases. Often they are irregularly placed and are stunted in their growth. Of the dental malformations, the most characteristic is the notching of the upper central permanent incisors.

3. The lymphatic glands, especially those of the neck, are enlarged, hard, and painless. Unlike the enlarged glands in scrofulous persons, they do not readily become swollen, soft, and caseous.

4. The periosteum, particularly that covering the long bones, presents swellings which are hard and usually painless.

5. Impairment of hearing, leading frequently to absolute deafness, is a common condition in these patients, and is due to the extension of nose and throat lesions into the middle ear.

It is rare to find all of these signs present in one individual; if a few of them are found, they form reliable evidence of the hereditary taint.

Fig. 226.—Notched teeth. (De Beck.)

Fig. 227.—Pegged teeth. (De Beck.)

Interstitial keratitis begins with photophobia, lacrimation, pericorneal injection, and blurring of vision. The cornea becomes hazy either at its periphery or at the centre; the haziness may be general or circumscribed. When the opacity extends, the cornea looks like a piece of ground glass. Under a magnifying lens it is seen to be studded with minute maculæ, which are deeply placed. Early in the history of the case vision is much reduced, and blood-vessels are found in both the deep and superficial layers of the cornea. As the vessels extend the ground-glass appearance is replaced by reddish "salmon patches" (Fig. 1, Plate XI). These may appear above, below, at either side, or they may involve the whole cornea. In this stage the cornea is softened and its curvature may be changed by pressure of the lids; photophobia and blepharospasm are intense, and fissures appear at the outer canthus. These symptoms continue with intermissions for weeks or months. In some cases years pass before the disease stops. The complications of interstitial keratitis are iritis, cyclitis, and chorioiditis, which generally appear early, but may not come until late. The second eye is always involved, with few exceptions; usually the disease has progressed to its height in the one before the other eye is affected. During the acute period of the inflammation

the cornea appears white, gray, or yellow according to the amount of infiltration present, and the condition may be mistaken for corneal abscess. The new vessels arising in interstitial keratitis spring from the deeply placed vessels of the sclera and project into the cornea in the form of tufts. These vessels grow into the membrane until the pupil is concealed. They may be small and invisible except through a corneal microscope or magnifying glass, or so large as to be readily seen by the naked eye.

Gradually the acute symptoms subside and the cornea begins to clear, the clearing beginning at the margin. In favorable cases the healing process lasts from nine to twelve months. Long after the disappearance of the attack the surgeon will know that such an eye has been affected by this disease, by reason of a peculiar lustreless appearance of the iris seen through a cornea that is slightly opaque. The anterior chamber is deeper than in the normal eye, and the ophthalmoscope shows the signs of a previous chorioidoretinitis. Close examination shows in the cornea the remains of old vessels.

PATHOLOGY. — Interstitial keratitis usually manifests itself at the corneoscleral margin, as an opacity in the deeper corneal layers, which gradually invades the cornea until the entire membrane is cloudy. If the infiltration is dense and the vascularization plentiful, the cornea becomes salmon colored. The cloudiness is accompanied by a small-cell infiltration, which may be very dense in the deeper layers. Besides this, new-formed vessels are found. The whole may resemble granulation tissue (Fuchs). The infiltration is also found in the ligamentum pectinatum, in the iris and the ciliary body, and sometimes in the sclera. Von Michel and others distinguish between a primary and a secondary parenchymatous keratitis. The opacity appears first at the limbus; and is complicated later by iritis, turbidity of the aqueous humor, and deposits on Descemet's membrane. Von Michel explains the disease as a consequence of syphilitic arteritis or hyalin degeneration of the pericorneal vascular loops.

He is opposed by Leber and von Hippel, who consider the affection as a secondary manifestation which is primarily an inflammation of the uvea. The anatomic findings of von Hippel, the infiltration in the deeper portions of the cornea, which embryologically is the uveal portion of the cornea, and the black patches which are in the periphery of the chorioid at the beginning of an interstitial keratitis, all support the latter view. Wagenmann produced a condition resembling this affection by cutting the two long and some of the short ciliary vessels. He concluded that the seat of the disease was to be sought in the chorioid.

DIAGNOSIS.—Attention to the symptoms described above will clear the diagnosis. The age of the patient and the condition of ocular tension will exclude primary glaucoma. The history of the case will serve to differentiate parenchymatous keratitis from recent infiltration due to trauma.

PROGNOSIS.—Interstitial keratitis is a chronic disease lasting from a few weeks to many months. In general terms it must be said that the

prognosis is favorable, since recovery of useful vision occurs in the great majority of cases. In scrofulous subjects the prognosis is not so favorable as in those of a purely syphilitic type. Recurrences of interstitial keratitis are rare and may come years after the primary attack. Restoration of normal vision is not to be expected in this disease, but the resulting opacity may be scarcely distinguishable. If improperly treated, the result may be disastrous through the formation of iritic adhesions, increased intra-ocular tension, etc. The duration of an attack of interstitial keratitis cannot be foretold. Early in the disease it is well to warn the patient that the vision will become much worse, while in the height of the attack he can be told that it will improve. In general, the prognosis is more favorable in adults than in children.

TREATMENT.—While some eminent observers hold that the disease is uninfluenced by treatment, the author believes such teaching to be pernicious and sometimes destructive. Although the therapeutic agents may not shorten the duration of the disease, it is not the less necessary that means should be employed to relieve pain and inflammation and keep the iris out of the way of harm. Both constitutional and local measures are to be used. Attention to the digestion, the taking of a reasonable amount of exercise, and the leading of an outdoor life will be in order. The internal use of mercurial preparations and bitter tonics is to be commended. Hutchinson's view of the value of mercury has been confirmed by many able clinicians. Codliver-oil, iron, and quinin can be used at the same time. If definite signs of hereditary syphilis are absent, it will nevertheless be wise to use mercury for several weeks or months. The remedy can be given by the mouth or preferably by inunction. Care must be taken to avoid ptyalism. Later in the case the iodids are serviceable. In weak cachectic subjects, mercury should be used sparingly and tonics should be exhibited freely. The syrup of the iodid of iron is a valuable remedy in these cases.

As regards local treatment, smoked glasses are to be used and atropin is to be instilled three times a day. The use of atropin must be continued long after the opacity renders the pupil invisible. It must be used freely, because the absorptive power of the cornea is diminished. The use of hot water is valuable in relieving pain. After the acute stage is over the daily application of an ointment of ammoniated mercury or the yellow oxid, combined with massage, will assist in clearing the opacity. Astringents and counter-irritants are to be avoided.

Vascular Keratitis (Pannus).—Of the forms of non-suppurative keratitis, this is, by far, the most frequent. Pannus is a small-cell infiltration containing newly formed vessels and involving the conjunctival (*i.e.*, superficial) layers of the cornea (Fig. 2, Plate X). The infiltration is in the deeper parts of the epithelial layer, and here there is a development of minute blood-vessels continuous with those of the limbus. The newly formed connective tissue is found chiefly between the epithelial and Bowman layers, but in some cases the process extends more deeply and involves the substantia

propria. It is proper to state that some authorities (Raehlmann, Nuel) have found the process in pannus to begin in a cellular infiltration beneath Bowman's membrane, which is lifted up, wrinkled, and finally perforated. Pannous tissue is thicker at the periphery of the cornea than at the centre; thus, at the periphery all the layers, down to that of Descemet, may be involved, while in the centre usually only the parts adjacent to Bowman's layer are affected. True trachoma bodies may be found in the corneal substance. Vascular keratitis exists in almost all cases of granular conjunctivitis, and

Fig. 228.—Trachoma with pannus. (Author.)

(Original drawing by Dr. R. W. Mills.)

is often found in phlyctenular kerato-conjunctivitis. The development of vascular keratitis leads to irregularities in the corneal surface, to great diminution of vision, and in some cases to projection of the softened cornea from intra-ocular pressure.

Symptoms.—Clinically it is necessary to divide pannus into several types, the appearance of the cornea varying from a slight vascular opacity to a condition comparable to raw flesh or a red rag. "In former times the adjective *tenuis* was used to indicate a thin layer of opacity, in which few vessels were present; *crassus,* a thicker and more advanced condition; *sar-*

carnatosus, a fleshy state of the cornea; while *siccus* was applied to a pannus that had undergone retrogressive changes" (Stephenson). Pannus generally is limited to that part of the cornea which is covered by the upper lid, but there are many exceptions to this. Thus, in the pannus of phlyctenular disease it is not uncommon to find broad, tongue-like vascular projections growing—one from above, the other from below—toward the centre of the cornea; and frequently, in severe granular conjunctivitis, the whole cornea becomes vascular. Pannus usually shows ulceration at some time in its history, but there are cases in which this is absent and the condition is one of hypernutrition. In typical pannus the upper third or half of the cornea is involved; numerous small, straight vessels, continuous with the posterior

Fig. 229.—Section of the cornea in pannus. (AUTHOR.)

(Original drawing by DR. CARL FISCH.)

1, Degenerated epithelium. 2, Area of round-cell infiltration. 3, Substantia propria. 4, Blood-vessels.

conjunctival system, are seen branching arborescently into a grayish opacity; and the epithelium is elevated and roughened. Vision is much reduced during the active period of the pannus, and later the sight in many cases is below the normal by reason of the irregular astigmatism and remaining opacities. When the pannus is thin, complete restoration will take place.

Pannus appears in the early stages of trachoma and almost never arises in the cicatricial stage. Relapses are common; they appear suddenly and seem to be provoked by meteorologic conditions, such as excessive humidity of the atmosphere or increased velocity of the wind. The signs of relapse are unmistakable: the conjunctiva becomes redder and thicker; the cornea, which possibly had cleared so that vessels were scarcely visible, becomes

studded with vessels; in two or three days small superficial ulcers appear; and pain, blepharospasm, photophobia, and lacrimation become prominent and annoying symptoms. Vision is rapidly reduced. A patient whose cornea was almost clear, in less than a week was reduced to a condition requiring the services of a guide.

DIAGNOSIS.—The diagnosis of pannus is easily made by inspection by the naked eye or with the aid of a magnifying glass. To distinguish between the granular, phlyctenular, and traumatic varieties of corneal vascularization is an easy matter, in view of the history of the case and the condition of the palpebral conjunctiva.

PROGNOSIS. — Granular pannus is an extremely persistent affection, the state of the cornea keeping pace with the condition of the conjunctiva. Generally the cornea clears, leaving useful vision; but in some cases pannus crassus lasts for years and destroys vision by causing permanent opacities. In other instances ulcers of the cornea accompanying pannus become infected, the cornea sloughs and is perforated, and the eye is left with anterior synechiæ and a staphyloma. In severe cases perforation is followed by panophthalmitis and loss of the eye. In many cases where pannus yields successfully to treatment, the cornea is left irregularly astigmatic. Recurrence of granular pannus is likely to take place at any time so long as the conjunctiva is granular.

The pannus accompanying phlyctenular disease generally yields promptly to treatment, leaving the cornea either unimpaired or the seat of a small cloud. Some cases, however, are followed by permanent opacities.

TREATMENT.—The treatment of pannus will depend on its cause and stage. In granular pannus, as a rule, little care is given to the pannus proper, the surgeon's attention being devoted to the conjunctiva. In the ordinary cases of trachoma treatment of the conjunctiva is followed by improvement in and disappearance of the pannus. In old cases, where, in spite of conjunctival treatment, the cornea remains vascular, it will be proper to employ remedies directed against the condition. There are a few cases of trachoma with ulcerating pannus which require paracentesis of the cornea. In such cases there is intense pain. Paracentesis is followed by relief of pain and general improvement in the eye. If an ulcer of the cornea is about to perforate, paracentesis should be resorted to, the instrument passing through normal tissue, and not through the floor of the ulcer. The pannus of phlyctenular disease usually clears up under the local application of the yellow oxid ointment and the use of proper constitutional remedies.

In pannus siccus good results sometimes follow massage with one of the mercurial ointments continued for a long period; but in many cases it will be necessary to use the irritant treatment by jequirity. A 5-per-cent. infusion of the seeds is brushed on to the eyelids and cornea, and the resulting conjunctivitis is intensified by repeating the application in twenty-four or forty-eight hours, as necessary. The remedy sets up an acute inflam-

mation, with enlargement of the vessels in the cornea, swelling of the conjunctiva, and the production of a grayish membrane on the affected tissues. There is some constitutional disturbance and an increase of the temperature. The pre-auricular and cervical glands may be swollen. The subsidence of jequirity-ophthalmia is followed in many cases by a clearing of the cornea. The remedy is not without danger. Rules for its use have been formulated by Nuel as follows: (1) the pannus should be complete, since non-vascularized portions may perforate; (2) the cornea of the second eye should present more or less pannus, the result of a contagious conjunctivitis; and (3) the palpebral conjunctiva should show a granular or cicatricial condition with slight discharge. Where there is an abundant discharge the use of jequirity is unsafe, since it causes too severe a reaction. If these precautions are not observed, the jequirity inflammation may produce ulceration of the cornea with perforation and loss of the eye. In any event suppurative dacryocystitis is sometimes caused.

In some cases of old rebellious pannus, where the cornea remains vascular after trachoma has been cured, the operation of periectomy, as described on page 301, is followed by improvement.

Keratitis Bullosa (Pemphigus of the Cornea; Keratitis Vesiculosa).—This rare variety of vesicular corneal inflammation occurs chiefly in middle-aged or elderly persons. It may be found in eyes which previously have been normal, but more often it occurs in those which are affected with glaucoma, iridocyclitis, or corneal cicatrix. A few cases are due to trauma, such as abrasion of the cornea by the finger-nail. The characteristic feature of the disease is the formation of a large vesicle, or bulla, which involves the outer part of the corneal surface and is tremulous. After a few days the vesicle ruptures, and coincidently there is great pain. The anterior wall of the vesicle, consisting of the epithelial layer (sometimes the deeper layers) of the cornea, is not shed, but remains *in situ*. After a variable time it is lifted up by fluid, bursts again, and this process is often repeated for many weeks. Ciliary injection, photophobia, lacrimation, increased tension, and pain are prominent symptoms during the development of the vesicle. Often these symptoms subside after the vesicle bursts. Fuchs explains the origin of corneal bullæ as follows: In glaucomatous eyes lymph-stasis causes an interstitial edema and results in lifting up the epithelial layer; if the fluid penetrates Bowman's layer, the latter also is lifted up.

PROGNOSIS in bullous keratitis is unfavorable. During the height of the disease the eye is painful, and vision is much reduced or entirely lost. After recovery the cornea remains opaque. There seems to be no tendency to the involvement of the second eye. Under the most skillful treatment the disease will continue for weeks or months.

TREATMENT should aim to relieve pain and irritation and reduce increased tension. The use of collyria of holocain and cocain gives only temporary relief. Removal of the anterior wall of the vesicle and the application of strong solutions of silver or burning with the galvanocautery may

be followed by improvement. If the intra-ocular tension is increased does not yield to arecolin or eserin, an iridectomy should be made. A tion should be given to the general health. In some cases tonics and septics can be used with benefit. If vision is lost, and recurring cro vesicles make the patient miserable, enucleation for the relief of pain be a justifiable operation.

Aspergillar Keratitis (Keratomycosis Aspergillina) is a variet corneal inflammation which is more common than would be supposed the limited literature. Probably locality has much to do with its freque one country physician living near St. Louis having met with seven within two years. The only case of the disease which the author has came from the same vicinity. The disease begins with great pain in the together with photophobia and lacrimation. After twenty-four or f eight hours a small black body is visible in the substance of the co

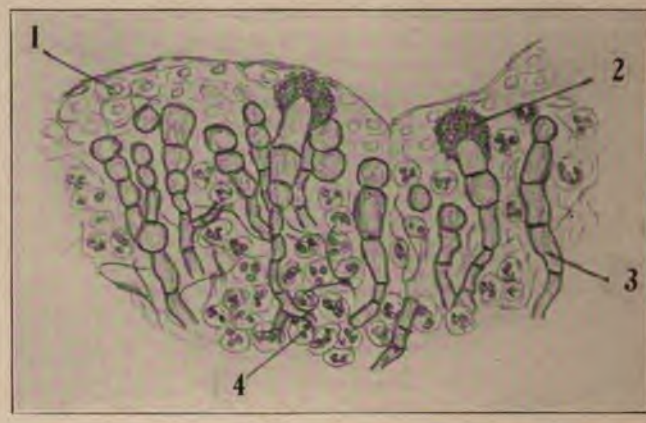


Fig. 230.—Microscopic section of growth in aspergillar keratitis. (AUTHO
(Original drawing by DR. CARL FISCH.)
1, Epithelium of the cornea. *2*, Sporangia. *3*, Mycelium. *4*, Pus-corpuscles.

The pain is constant and increasing, and is out of all proportion to resulting from the lodgment of a foreign body in the cornea. After secting the black body from the cornea the patient makes a rapid reco If the case is not treated early by removal of the small black body superficial layers of the cornea slough, leaving an ulcer which lasts after the surgeon removes the aspergillar mass. This is of a black or brown color, and may be mistaken for an ordinary foreign body. The is rounded and soft. Microscopic examination shows its nature. Fa to recognize the condition and remove the aspergillar mass may resu loss of the eye through corneal sloughing, perforation, and panoph mitis. In the few cases of keratomycosis aspergillina in which cultures been made only *Aspergillus fumigatus* has been found.

Wicherkiewicz saw a case of keratitis produced by *Penicillium glau* and Wolfner met with one in which *Mucor corymbifer* was present.

Malarial Keratitis (Dendritic Keratitis; Keratitis Dendritica Exulcerans Mycotica).—Although in a strict pathologic sense it is incorrect to speak of a malarial keratitis, since the *Plasmodium malariæ* cannot produce an acute inflammation, yet from a clinical point of view it is wise to retain the term. The disease is a superficial keratitis, attended by a dendritic ulceration, occurring in persons sick with malaria. It is found chiefly between the ages of twenty and fifty years, is more common in males than in females, and is seen more frequently in the summer and fall than in the winter and spring. The ocular disturbance, which is usually preceded by intermittent fever, begins with pain, photophobia, lacrimation, and the sensation of a foreign body in the conjunctiva. In some cases the disease begins with supra-orbital neuralgia. Within a few hours after the advent of these symptoms, numerous small, rounded, bead-like elevations are seen in the cornea; and a day later the elevated spots disappear, leaving superficial ulcerations of irregular shape (Fig. 5, Plate XI). The process extends, small, branching furrows running off from the initial lesion. The furrows are of a bluish-gray color. The ulcers do not penetrate deeper than Bowman's layer. The cornea is anesthetic, tension is normal, and hypopyon

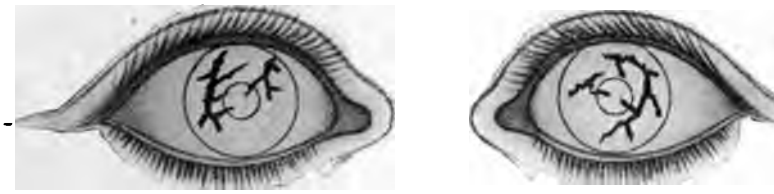


Fig. 231.—Malarial keratitis. (Kipp.)

rarely occurs. Pain in and around the eye is severe and continues until the process of ulceration has been checked. Iritis is a rare complication. The duration of the disease varies from two or three weeks to as many months. The resulting opacities are not usually dense, but can be seen by oblique illumination many months after cure. Recurrence of the keratitis may appear with each recurring attack of malarial fever.

Etiology.—Although Kipp and Hotz, who were among the first to recognize this disease, formerly held it to be pathognomonic of malaria, further observation has shown this view to be erroneous. Dendritic keratitis sometimes occurs in healthy persons. It has been observed in scrofulous and tubercular subjects. Kipp now believes that malaria is the cause of 90 per cent. of cases of dendritic keratitis. Noyes held that, while this peculiar form of keratitis could co-exist with malaria, it was essentially a mycotic disease. Knapp has seen superficial keratitis as a symptom of acquired syphilis.

Prognosis.—The prognosis of dendritic keratitis is favorable.

Treatment.—The use of atropin, of protective spectacles, and the occasional instillation of a 1-per-cent. strength solution of holocain, to

relieve pain, will be advisable. If the disease extends, all of the furrows and their ramifications should be thoroughly scraped under local anesthesia and a solution of bichlorid of mercury (1 to 2000) should be used to flush the conjunctiva. This treatment usually checks the process, and the use of the galvanocautery will rarely be necessary. The general treatment is of great importance in these cases. Often the keratitis will prove rebellious to all local measures and will yield readily after a prolonged course of anti-malarial medication.

Filamentary Keratitis.—This unusual condition, which may follow corneal wounds or vesicles, is characterized by the appearance of small globules attached to the cornea by a twisted pedicle or filament. These

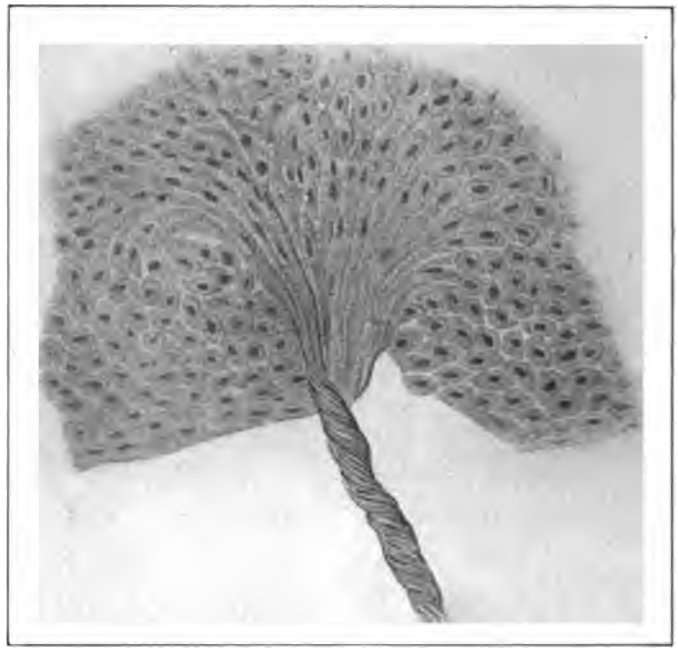


Fig. 232.—Filamentary keratitis. (HESS.)

apparent vesicles are small, the largest having a diameter of 1 to 1.5 millimetres. From time to time a new eruption appears, and thus the disease may continue for months or years. The disease, which occurs chiefly in persons of advanced age, produces the usual signs of keratitis. When examined microscopically the filaments are seen to consist of granular masses attached by a rope of epithelium, thick at its corneal attachment and pointed at its extremity. The condition, which originates in the corneal epithelium (Hess), is likely to recur, successive crops of vesicles and filaments being produced. The disease sometimes occurs in chronic ulcers and in the keratitis bullosa of absolute glaucoma (Hess). Filamentary keratitis may be confounded with herpetic keratitis. Removal of the filaments by curettement is followed by relief, but recurrences are frequent. In these cases the

use of a 2-per-cent. strength solution of ammonium chlorid is said to be beneficial.

Keratitis Punctata (Descemetitis; Aquo-capsulitis; Serous Iritis; Serous Cyclitis).—This affection, which by some writers is discussed under corneal diseases, will be considered in Chapter XI under the name "Serous Cyclitis," because recent investigations have shown that it depends on alterations in the glands of the ciliary body. The characteristic feature of keratitis punctata is the presence, on the posterior surface of the cornea, of numerous dots of fibrin, which usually are disposed in the form of a triangle whose base is downward toward the corneoscleral junction *(keratitis punctata profunda)*.

Bruns contends that there cannot be such a disease as serous iritis, since descemetitis (punctate keratitis), the symptom on which the diagnosis of serous iritis was long made to rest, is most frequently the principal symptom of acute plastic chorioiditis. In the absence of an ophthalmoscopic examination the chorioidal disease is overlooked.

Fig. 233.—Keratitis punctata. (AUTHOR.)

(Original drawing by DR. R. W. MILLS.)

Superficial Punctate Keratitis (Keratitis Subepithelialis Centralis; Nummular Keratitis; Relapsing Herpes of the Cornea; Macular, or Nodular, Keratitis).—A form of punctate keratitis, located in the superficial layers of the cornea, and occurring in young subjects during an attack of acute catarrhal conjunctivitis, has been named *keratitis punctata superficialis* by Fuchs. The cornea presents minute gray dots, often arranged in groups or rows, scattered irregularly over the cornea or massed in the centre. They may be present coincident with the conjunctival inflammation or may follow it by days or weeks. The spots cause the epithelium to bulge and give the cornea a cloudy look. Often the spots remain unchanged for months and gradually disappear. Like herpes of the cornea,

this disease sometimes develops simultaneously with an acute catarrhal inflammation of the respiratory tract, but it is distinguished from herpes by the absence of vesicles. The attack usually begins with symptoms of conjunctivitis, with profuse lacrimation, and with catarrh of the air-passages. In two or three days numerous gray or yellowish-green spots appear beneath Bowman's layer; the cornea becomes hazy, and often gray radiating lines are present. The spots, which are one millimetre or less in diameter, should be looked for with a magnifying glass. Loss of vision in this disease will be commensurate with the number and location of the spots. If they are numerous and occupy the pupillary area, vision will be much reduced. One or both eyes may be involved. Although some authors state that iritis and hypopyon never are seen in this affection, it will be well to bear in mind the possibility of their occurrence.

ETIOLOGY.—As regards the etiology of this disease, Nuel has found it chiefly among subjects between twenty and thirty-five years of age, who had been exposed to intense cold. Microscopic study shows the disease to be due to an ill-identified coccus—according to Valude, the staphylococcus.

Fig. 234.—Appearance of the cornea in punctate keratitis. (KNIES.)
A, Keratitis punctata superficialis (Fuchs). *B*, Opacities found in parenchymatous keratitis.

PATHOLOGY.—Nuel has found the edema of this disease to be located in the anterior corneal lamellæ. The spots are due to filaments of fibrin undergoing a hyalin change; the filaments are disposed in spiral form and are situated between the corneal lamellæ. They form the spots described above. At the level of these spots the corneal epithelium forms projections, which are converted into lacunæ. Micrococci are found in the swollen epithelium. There is an absence of invasion by migratory cells.

PROGNOSIS.—The prognosis of this form of keratitis is favorable. Although the course of the disease may be prolonged, the spots gradually disappear by resorption.

TREATMENT.—Irritants and astringents are out of order in this disease. Atropin, dark glasses, and attention to the general health will comprise the therapeutic measures to be used. If increased tension occurs, a paracentesis of the cornea should be made.

Ribbon-shaped Corneal Opacity (Calcareous Keratitis; Primary, or Zonular, Opacity of the Cornea; Transverse Film of the Cornea; Keratitis Trophica; Keratite en Bandelette).—This unusual form of keratitis may be congenital or acquired. As a congenital condition it is of exceedingly

rare occurrence. In the acquired form it is present in two distinct clinical types: In the one the interpalpebral part of the cornea, in eyes which were previously normal, becomes opaque, owing to the development of a smooth *subepithelial opacity*. In the other type an oval transverse band of opacity develops in eyes which have long been blind from iridocyclitis, sympathetic ophthalmitis, or glaucoma. This form of opacity produces a roughening of the cornea and occurs chiefly in the lower third of the tunic. Since this is the part of the eye which is exposed when the globe is rolled upward, as in sleep, the condition has been thought to be due to imperfect closure of the lids during sleep or in the course of exhausting disease.

In the first type the local change consists in a deposit of lime salts beneath the epithelium, which is unaffected. The opacity is sharply defined while the remaining cornea is clear. The opacity forms a gray area, three to five millimetres in width, passing across the cornea below the centre of the pupil. Often there is an oval opacity at the outer and another at the inner part of the cornea, separated from the limbus by clear corneal tissue.

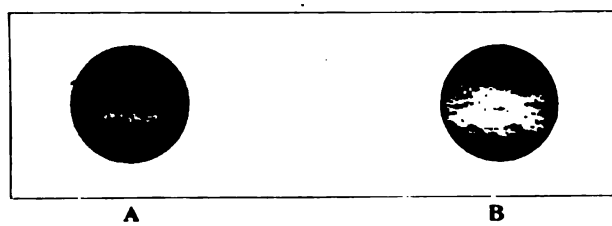


Fig. 235.—Appearance of the cornea in ribbon-shaped opacity. (KNIES.)

A, In an early stage. *B*, In a late stage.

The two oval masses are connected by a bridge through which the pupil can be seen.

Usually the process requires years for its development, but Poulett Wells has described a case of marked zonular opacity of the cornea which developed in a few months. Some authors have attributed these opacities to an excess of uric acid in the blood, while others think them due to external injurious influences. Fuchs saw transverse films in both eyes of a physician who for many years had blown calomel into them, and Topolanski met with the disease in hat-makers whose eyes were daily irritated by flying pieces of hair.

A corneal disease, which is similar to ribbon-shaped opacity, is found among persons engaged in the manufacture of anilin dyes and of naphthalin. The long-continued irritation of the fumes produces a brown or gray superficial opacity of the cornea. The affected zone corresponds to the interpalpebral cleft. The opacity, which is located in the epithelial and superficial corneal layers, slowly disappears after the patient changes his occupation.

In the period of repair the frequent use of hot water and the local application of an ointment of ammoniated mercury or yellow oxid, or the insufflation of a powder of boric acid or aristol will be in order. The application of tincture of iodin has been recommended. In rebellious cases recourse can be had to a mild use of the galvanocautery. The internal treatment is of importance. The respiratory organs should be given the necessary attention. In cases characterized by periodic recurrences the use of a saline purge, followed by quinin and tonics, will be of value.

Marginal Keratitis (Keratitis Ulcerativa Marginalis).—Ulceration of the cornea occurring at the corneoscleral margin may assume several forms. Under the name *keratitis ulcerativa marginalis*, W. A. Martin, of San Francisco, has described cases of acute corneal disease occurring in adults and characterized by the following features: The initial symptoms are pericorneal injection and photophobia, followed by the development of a row of papillæ in the cornea about the line of the arcus senilis. These papillæ become confluent, break down, and form a line of narrow ulcers which may

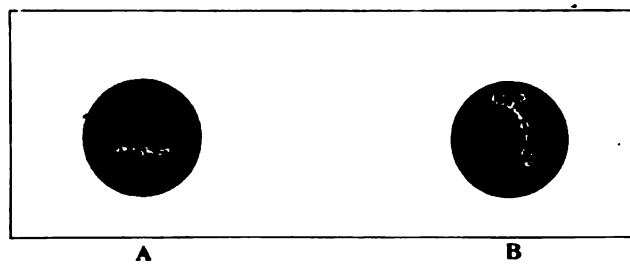


Fig. 236.—Appearance of the cornea in herpes febrilis. (KNIES.)

A, Early stage of herpes. *B*, The same eye four days later.

almost encircle the cornea. Separating the ulcerated spots from the limbus is a zone of transparent corneal tissue. The disease does not encroach upon the centre of the cornea. Under proper treatment healing occurs rapidly. In some cases, as soon as the ulcerated spots heal, others occur farther on, until the whole cornea has been traversed. Under daily use of a mild bichlorid solution and calomel insufflations healing occurs without scarring. Unlike the catarrhal ulcers described by Fuchs, keratitis ulcerativa marginalis is a primary disease.

A different type of marginal keratitis is that of Fuchs: *keratitis marginalis profunda*, which belongs to the class of parenchymatous or interstitial corneal inflammations. Here the process is a chronic one, and the transparent zone adjoining the corneoscleral junction is obliterated. It is a rare disease, occurring in old people and usually is limited to one eye. Grayish or yellowish-gray opacities, forming at the periphery, project into the cornea for two to three millimetres. The opaque zone involves generally one-third to one-half of the circumference. Irritative symptoms disappear in ten to fourteen days, but a permanent gray opacity remains and

resembles arcus senilis. It is distinguished from the latter by the absence of a zone of clear corneal tissue. Owing to the location of the lesions in this disease, no damage is done to vision.

TREATMENT.—In the treatment of marginal keratitis the surgeon is to be guided by general principles and common-sense. Atropin and holocain are to be used cautiously to relieve pain and photophobia, and a bichlorid solution is to be applied two or three times daily. If pain is violent, use can be made of dionin and cocain (dionin, gr. ij; cocain, gr. ij; water, 3iv. M. Sig.: One drop in the affected eye five or six times a day). Insufflations of calomel are valuable in marginal ulcers. If the ulcer extends in depth it will be necessary to apply the curette or electrocautery, while eserin or arecolin is used to contract the pupil.

Striped Keratitis (Striate Keratitis).—This name has been applied to two different conditions which occasionally occur after cataract extraction or trauma. In both there is an opacity which is located in the posterior layers of the cornea. In true striped keratitis there is noticed, at the first

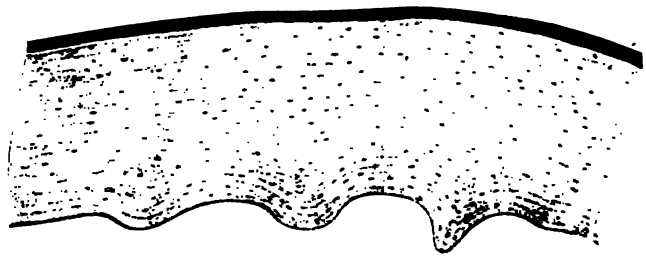


Fig. 237.—Section of the cornea in striate keratitis. (HESS.)

The folding of the posterior layers of the cornea is shown in the lower part of the figure.

or second dressing after cataract extraction, the presence of fine straight lines of opacity, one-half to one millimetre in width, which converge toward the wound. In some cases the lines cross, forming panel figures. Several theories have been advanced to account for the phenomenon. Beck and Recklinghausen attributed it to dilation of lymph-spaces; Alt, to infiltration of large nerve-canals; but the view now generally accepted is that of Carl Hess and others, viz.: that this peculiar striate opacity depends on the folding of Descemet's membrane, resulting either from the shrinking of the cornea by cicatrization or from unequal swelling of the corneal tissue by infiltration. However produced, the condition generally disappears within a few days. Striped keratitis is occasionally seen after corneal injuries or inflammation.

The other type of opacity, which is common after cataract extraction, is in the form of permanent white opacities, which are due to the action of chemicals. It is often seen in the practice of those surgeons who irrigate the anterior chamber with solutions of bichlorid of mercury. These opaque spots materially reduce vision and cannot be removed.

Disc-like Keratitis (Keratitis Disciformis).—A type of keratitis, which somewhat resembles ulcus serpens, is known as keratitis disciformis. It is characterized by the development, in the middle layers of the cornea, of a gray, discoid opacity. The disc is separated from the transparent peripheral portion of the cornea by a grayish border. In the middle of the disc is generally a small spot of deep infiltration. Surrounding this spot are curvilinear lines. Disciform keratitis does not become yellow and does not lead to corneal necrosis. It is only in exceptional cases that it leads to a loss of substance in a circumscribed area. Inflammatory symptoms are not marked. Hypopyon is either absent or is of small extent. The superficial corneal layers are generally unaffected, but, in the course of the disease, small ulcers may appear. This type of corneal inflammation generally lasts several months and leaves a cloud. Fuchs attributes it to the infection which follows a small epithelial lesion, and which is not sufficiently virulent to cause a serpiginous ulcer. Disc-like keratitis is to be distinguished from the deep and from the annular forms of corneal inflammation. Atropin locally and proper internal medication will comprise the treatment of this disease.

Grill-like Keratitis (Gitterförmige Keratitis; Kératite Quadrillée) has been described by Haab. The cornea presents a central opacity which, by transmitted light, shows forked lines with points scattered through them, the whole resembling latticework. Darier believes that it is due to an infiltration of leucocytes into the corneal spaces. He states that the disease improves rapidly under subconjunctival injections of sodium chlorid or under instillations of dionin.

MISCELLANEOUS DISEASES OF THE CORNEA.

Discoloration of the Cornea with Blood-pigment (Blood-staining of the Cornea).—This rare condition, which involves some difficult points in diagnosis, has been studied by Vossius, Treacher Collins, Lawford, Weeks, Griffith, and others. In a case of blood-staining of the cornea there is first a blood-clot in the anterior chamber, due usually to trauma or operation, although the hemorrhage may have occurred spontaneously in old cases of retinal detachment. In nine of seventeen cases mentioned by Collins the tension was increased; in six others it was normal or minus. The phenomenon may occur at any age. In the author's case, a man, aged 35 years, was struck on the right eye by a piece of steel. One month later an operation (probably an iridectomy) was immediately followed by hemorrhage into the anterior chamber and blood-staining of the cornea. Two years later the appearance of the eye was that represented in Fig. 3, Plate XI. The entire cornea was of a brick-dust color. In most of the cases heretofore reported a narrow peripheral ring, one to one and one-half millimetres wide, has remained clear and uncolored. This condition may be mistaken for an anterior dislocation of the lens. As regards the nature of the pigment

which is distributed through the cornea, the microspectroscopic and chemic examinations of Collins show that the discoloration is due mainly to crystals of hematoïdin, with or without hemosiderin, which enter the cornea in solution in blood diffused through Descemet's membrane. After a period varying from two to many years, the discoloration disappears, the periphery being the first to clear. Treatment of this condition seems to be useless, although alteratives may be tried.

As regards diagnosis, it will be necessary to distinguish between hemorrhage into the anterior chamber, forward dislocation of the lens, and blood-staining of the cornea. When the whole cornea is stained it cannot be distinguished from distension of the anterior chamber with blood, but if the peripheral clear zone is present, the distinction can be made. When the central part of the cornea is of a rusty-brown color from blood-staining, and the periphery is clear, the condition so much resembles that of an

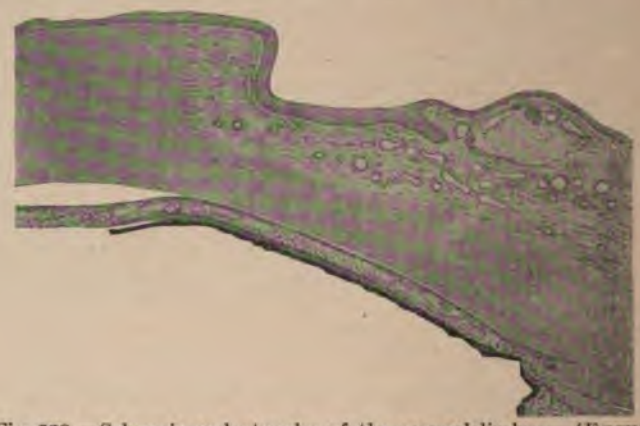


Fig. 238.—Sclerosis and atrophy of the corneal limbus. (FUCHS.)

The eye was glaucomatous, which accounts for the adhesion of the iris to the posterior surface of the cornea. Numerous blood-vessels are present in the atrophic and sclerosed limbus.

amber-colored lens, dislocated forward, that the best observers have been in doubt. It would seem that focal illumination ought to be of value in making the diagnosis. In the author's case focal illumination showed clearly the presence of wavy reddish lines in the cornea. Probably these were distended lymph-channels.

Sclerosis and Atrophy of the Limbus.—Fuchs has recently described a rare condition in which a groove-like depression circumscribes the periphery of the cornea without the occurrence of ulceration. The disease, which is found chiefly in elderly persons, is a senile change ingrafted upon a concomitant arcus senilis. There is a hyalin deposit in the superficial corneal layers. Some of the hyalin masses may be visible in the living eye, examined under a loupe, appearing as whitish points. Ectasia of the cornea has been present in some of these cases. There is no known treatment for the condition.

Siderosis of the Cornea.—In this condition opaque spots of a rust-brown color form in the cornea. The author has observed one such case of corneal staining due to the lodgment of a piece of steel in the lens.

Xanthelasma of the Cornea.—This is a degeneration of the cornea and is characterized by its yellow color. It follows injuries attended with shrinking of the globe and often exists with a calcareous deposit in the cornea. Such eyes are usually sightless. They are prone to occasional attacks of inflammation and may be the cause of sympathetic ophthalmitis. There is no particular treatment for the condition. If indicated, an enucleation or a Mules operation should be performed.

Tuberculosis of the Cornea.—The cornea, like the lens, is much less liable to tuberculosis than are the other ocular structures. The disease may be primary or secondary to tuberculosis of the conjunctiva or iris. Panas has recorded a case of primary corneal tuberculosis in a woman of 30 years, who had severe pain in the right eye and presented diffuse interstitial opacity of the upper segment of the cornea. Two weeks later the centre of the cornea showed yellowish nodules resembling miliary tubercles. These coalesced and formed an ulcer with yellow base and irregular margins. Eugene Smith, of Detroit, has observed a case of primary corneal tuberculosis. Three tubercular foci were present in the posterior part of the substantia propria without ulceration of the cornea and without implication of Descemet's membrane.

The diagnosis of corneal tuberculosis rests on the finding of tubercle bacilli in scrapings from the cornea. The treatment is the application of the electrocautery.

Lepra of the Cornea.—Keratitis occurring in lepers is not specific, but is generally due to traumatism. Ulceration, which is most frequent in the lower limbus, is rarely deep or acutely septic and generally heals under mild treatment with temporary closure of the eyelids. The infiltration in lepra of the cornea begins peripherally. There may be a small dotted, yellowish deposit; or there may be a distinct nodule involving the cornea, sclerotic, and iris simultaneously. Both eyes may be symmetrically involved. The infiltration consists partly of bacilli, but chiefly of cells in a brown state of granular degeneration (Neve). The process may extend and involve the iris and deeper ocular structures.

PROTRUSIONS OF THE CORNEA.

Protrusions of the cornea comprise staphyloma, kerectasia, keratoconus, and keratoglobus. The first and second of these originate in inflammatory, and the third and fourth in non-inflammatory processes.

Staphyloma of the Cornea.—This is a condition in which a corneal scar with adherent iris bulges outward (Fig. 4, Plate XI). It is total when the entire cornea is involved, partial when only a part is concerned, or racemose when perforation of the cornea has occurred at several points.

Staphyloma may have a spheric or a conic shape. It may be limited to the cornea or involve also the ciliary region. The alteration is brought about by intra-ocular pressure acting on a tissue which is softened by inflammatory action. Intra-ocular pressure in staphyloma is increased in one of two ways: if, as often happens, the iris is fastened to the posterior corneal surface, the anterior chamber is abolished and increased tension occurs from closure of the drainage outlet; in other cases it is assumed that, as the staphyloma increases, tension on the adherent iris causes irritation of the ciliary glands with increased secretion of the aqueous humor. Having attained a certain size, a staphyloma may remain stationary or it may further extend and rupture. The rupture closes only to reopen when tension rises. After repeated ruptures infection may take place and the eye may be lost by panophthalmitis. Although staphyloma may follow corneal inflammation, or may be present as a congenital condition without perforation, it is the rule that it follows perforation with iris-prolapse. If an iris-prolapse heals with the formation of a flat cicatrix, the result is favorable to the integrity of the

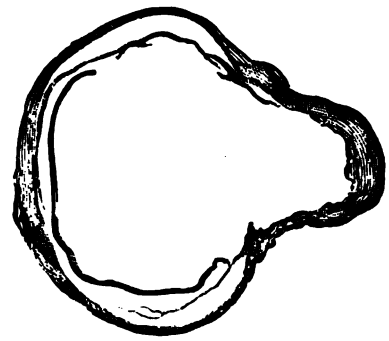


Fig. 239.—Staphyloma of the cornea. (Author.)

globe, although vision is much reduced; but if the protruding iris becomes covered with cicatricial tissue, which is too weak to resist intra-ocular pressure, the ectatic cicatrix forms a staphyloma. If the whole cornea is involved in the ectasia, we speak of *total staphyloma;* if only a part bulges, the staphyloma is *partial.* If all the corneal layers down to the posterior elastic lamina are destroyed, and this protrudes as a small bladder, the name *keratocele* is applied to the condition. After a corneal wound, or following upon a perforating ulcer, a minute opening may remain for a considerable time; or a keratocele may break from time to time. In either event the term *fistula of the cornea* is used to describe the condition. When a large part of the cornea is destroyed, the exposed area is covered with a transparent layer of lymph, which deludes the patient into the belief that he will retain useful vision. Soon, however, the new tissue becomes opaque, and vision is reduced to quantitative perception of light.

Symptoms.—Early in the history of staphyloma pain, photophobia, lacrimation, and redness are present. The opaque cornea is seen to bulge.

Gradually the inflammatory symptoms subside and the staphyloma appears as a protruding mass, which may be so extensive as not to be covered by the eyelids. In this event the apex becomes irritated, or it may become dry and resemble skin. A corneal staphyloma forms an opaque mass of whitish, bluish, grayish color, traversed by a few large vessels. Often in old cases there is a calcareous deposit in the apex of the mass. Hyalin degeneration is not uncommon in old cases, and keloid sometimes grows from a thickened staphylomatous cornea. If the protruding cornea is thin and translucent,—*i.e.*, the process is of recent date,—it will be possible to discern the outlines of the iris. Tension in old staphylomata may be normal, but, whether normal or increased, it leads sooner or later to rupture of the staphyloma or to excavation of the nerve-head. The tendency of a corneal protrusion is to become large and involve the entire globe, leading to general distension of the eyeball. Vision may be retained sufficiently to permit the patient to count fingers and find his way about, but usually it is limited to perception of light. Suppurative inflammation may occur, owing to exposure of the protruding mass to the irritating effects of air and dust. In such a case there will be great pain and marked inflammatory symptoms. In quiet staphylomata there is often considerable pain from pressure of scar-tissue upon nerve-filaments or from the presence of irido-cyclitis.

Diagnosis.—Staphyloma can be distinguished from corneal opacities by the presence of the protrusion. New growths appearing on the cornea and springing from the corneoscleral region externally, or intra-ocular tumors which have broken through the ocular shell, may cause the inexperienced observer to be in doubt, but the history of the case and close attention to the appearance of the eye will clear the diagnosis.

Prognosis.—Corneal staphyloma must always be regarded as a serious condition. Its prevention is of greater importance than its treatment.

Treatment.—The prevention of staphyloma concerns the proper treatment of iris-prolapse. To secure the production of a flat scar is of the utmost importance in cases of iris-prolapse (see page 316). When this result has been attained, means should be employed to prevent bulging of the scar. They include the use of such measures as the compress bandage, the application of a mild miotic, and in suitable cases the operation of iridectomy—all for the purpose of reducing or limiting the effect of intra-ocular tension upon a softened tissue. Küchler, in hopeless cases of sloughing cornea, with protrusion of iris and cicatricial tissue, in order to prevent staphyloma, passed a cataract-knife horizontally through the cornea, opened the capsule, and delivered the lens. This operation prevents staphyloma, leaves the globe of normal size, and does not increase the danger of panophthalmitis.

Conspicuous staphylomata that cause loss of useful vision require enucleation of the eye, or, preferably, a Mules or Hall operation. Excision of the staphyloma—an operation much practiced by de Wecker, Critchett, and others of the older ophthalmologists—is not to be consid-

ered, since such a procedure not rarely results in sympathetic ophthalmitis or causes panophthalmitis. Particularly dangerous is Critchett's operation, in which needles are passed through the base of the staphyloma to prevent loss of the vitreous humor.

Keratoconus (Conic Cornea; Staphyloma Pellucidum).—In this condition the curvature of the cornea is changed from an ellipsoid to an hyperboloid of revolution. It is due to a disturbance between the resisting power of the cornea and intra-ocular tension, and ordinarily appears at or about the time of puberty, but may be congenital. Generally both eyes are affected. It is more frequent in females than in males. The apex, which is usually transparent, may show an opaque area. The protrusion may be slight or marked. In the former case the diagnosis of keratoconus will

Fig. 240.—Keratoconus. (AUTHOR.)

(Original drawing by DR. R. W. MILLS.)

rest on the skiascopic test, which shows a triangular area of light, the base of the triangle resting on the margin of the iris and the apex corresponding to the apex of the corneal cone. The ophthalmometer can be used satisfactorily in the diagnosis of keratoconus. The mires are much distorted. That the deformity is not due to old corneal lesions can be shown by the absence of nebulæ. If Placido's disc is used in keratoconus, the circles are distorted. To the ophthalmoscope the details of the fundus are distorted, and a circle-shaped shadow is present. These effects are due to the high degree of corneal astigmatism produced by the projection. In marked cases of keratoconus the diagnosis can be made without instrumental aid by placing the patient alongside a window and viewing the cornea laterally.

The subjective symptoms of keratoconus include near-sight, polyopia, and an indefinite uncomfortable sensation in the eyes and head. Inflam-

matory symptoms are usually absent. Relief is sought for defective vision, which often is reduced to one-thirtieth or one-fiftieth of the normal. Concave glasses give some improvement for a time. Clear vision cannot be obtained by concave spheres, since the cornea is not spheric, but conic. If the apex of the cone becomes infiltrated, vision is made much worse.

PATHOLOGY.—The pathology of conic cornea is not understood. It is supposed to depend on a diseased condition of the uveal tract.

PROGNOSIS.—The prognosis will depend on the condition at the time the patient applies for treatment. If the apex of the cone is clear, the prognosis may be considered to be favorable. In some cases the protrusion stops at a certain point.

TREATMENT.—In the early stages of conic cornea, when the disease is progressive, as is manifested by pain in and around the eye, the prolonged use of a mydriatic and bandage will be beneficial. Some surgeons, while continuing the use of the bandage, alternate mydriatics and miotics. After headaches and tenderness of the globe have disappeared, the refraction is

Fig. 241.—Keratoglobus. (After DEMOURS.)

to be corrected. Here crossed cylinders will often be beneficial. Raehlmann proposed to correct these cases by hyperboloid lenses made in Rathenow, but these have been discarded, because their use is limited. They are of value when their apices coincide with those of the corneal cones, and this obtains in only one position of the eye. Stenopaic spectacles are sometimes of benefit in cases of keratoconus, and are used by Snellen.

The surgical treatment of conic cornea includes many procedures, of which the most valuable is the careful use of the galvanocautery, as is explained in the latter part of this chapter.

Keratoglobus (Cornea Globosa; Megalocornea).—By the older authors hydrophthalmos was confounded with keratoglobus, but Horner, Pflüger, and others have shown that the latter disease has nothing in common with buphthalmos. In keratoglobus the cornea presents a globular form, and is enlarged and clear. The iris also is enlarged. In two cases seen by Fick the eyes were hypermetropic, and in each visual acuity, tension, and fundus were normal. The affection is usually bilateral, and is not amenable to treatment.

Keratectasia (Keratocele).—This is the name applied to a bulging forward of the deeper corneal layers, without perforation and without involvement of the iris. The condition is made possible by ulceration of a cornea which is distended by intra-ocular pressure. In rare instances it is sequent to recurrent attacks of marginal keratitis. Keratectasia is usually partial in extent and irregular in form, but sometimes it is regular and occupies the whole cornea. The latter cases usually are due to pannus of the substantia propria or to parenchymatous keratitis with softening of the cornea. If the cornea is ulcerated down to the membrane of Descemet, this layer is pushed forward in the form of a hernia through the perforation and the condition is termed keratocele. It appears as a transparent vesicle surrounded by an opaque cicatricial ring. Keratectasia is usually distinguishable from keratoconus and keratoglobus by the fact that the bulging portion of the cornea is opaque. A few cases of corneal ectasia have been observed following marginal keratitis, in which the ectasia was composed of apparently normal corneal tissue.

PROGNOSIS.—The prognosis in keratectasia is unfavorable. If the condition is detected early and is checked by treatment, much damage is done to vision, owing to the opacity and the irregular bulging of the cornea. In fully developed keratectasia treatment is powerless.

TREATMENT.—In the formative stage repeated punctures of the cornea with a cataract-knife or small keratome should be practiced. The evacuation of aqueous humor is followed by temporary decrease in intra-ocular tension. This treatment should be followed by the application of a compress bandage. After the process has reached maturity, if the keratectasia is small, it can be destroyed by the galvanocautery or punctured with a keratome, and in its place a flat cicatrix will form. Here also the compress bandage will be of value. The obstacles to excision of the affected area and the grafting therein of part of the cornea of one of the lower animals are at present so great as to make this operation of doubtful utility.

Fistula of the Cornea (Fistulous Staphyloma).—This name is applied to two conditions. A *true corneal fistula* exists when, after a perforating wound or ulcer, a prolapse of iris occurs which is not large enough to close the tubular opening. Although the lens comes forward after the escape of aqueous humor and temporarily blocks the opening, healing does not occur, because a piece of the iris lies in the wound. In true corneal fistula there is a downgrowth of epithelium which also interferes with the healing process.

A *spurious fistula* exists when a small corneal staphyloma repeatedly ruptures under the influence of intra-ocular pressure of either normal or increased amount. Microscopic examination shows an absence of epithelial downgrowth. The iris may or may not be incarcerated in the wound. In a case of this kind following a corneal ulcer, occurring in the author's clinic, the patient was in the habit of opening the apex of the small staphyloma with a needle, whenever increased intra-ocular tension produced

pain in the eye. The evacuation of aqueous humor was followed by several days of relief. As usually happens in corneal fistula, the case ended in an enucleation. Histologic examination showed an absence of epithelial lining.

These definitions are based on pathologic studies. To distinguish the conditions clinically is often impossible.

Clinically, a fistula of the cornea exists as a weak spot which alternates between closure and patency. Its site is indicated by the presence of a small, black point from which the aqueous humor seeps. When located near the centre of the cornea, the black point is absent and the area surrounding the fistula is composed of scar-tissue.

A corneal fistula may follow a wound or be a sequel of ulcerative keratitis. It may be located in any part of the cornea. When the cicatrix breaks, the aqueous humor is lost; the lens comes forward and may touch the borders of the fistula, causing an anterior polar opacity. The eye becomes soft and may eventually be lost by infection or by atrophy of the globe; or the closing of the fistula may be followed by glaucomatous symptoms.

Fig. 242.—Fistulous ulcer of the cornea. (WÜRDEMANN.)

PROGNOSIS.—In brief, the prognosis of corneal fistula is unfavorable, the eye, as a rule, being destroyed by chronic iridocyclitis, by panophthalmitis, or by secondary glaucoma.

TREATMENT.—This admits naturally of division into (1) medical or non-operative and (2) surgical treatment.

1. The non-operative measures include rest in bed and the use of miotics (proposed by Zehender in 1868), for the purpose of reducing intra-ocular tension, and the application of a compress bandage to give support to the weakened tissues. Under this plan there is often an improvement. The anterior chamber re-forms and the surgeon may expect a cure. Often he will be disappointed. The intra-ocular tension rises, the weakened tissues rupture again, and the anterior chamber is abolished. Thus, the eye alternates between a condition of abnormal softness, due to an open fistula, and a state of gradual increase of intra-ocular pressure until rupture occurs. Such eyes often pass into a condition of chronic iridocyclitis with softening, or they may show panophthalmitis. As a rule, non-operative treatment is

of value only in so far as it favors the restoration of the anterior chamber and renders the performance of iridectomy possible. The miotic to be employed should be arecolin, which is less irritating than eserin or pilocarpin. It is to be employed in a $^1/_2$- to 1-per-cent. strength solution, 2 or 3 drops being instilled into the conjunctival sac twice or thrice a day. If, under the use of the miotic and the bandage, the condition improves, nothing further will be necessary. If, however, the weakened cornea again breaks, as frequently occurs, only surgical measures can be looked to.

2. Surgical measures for the cure of corneal fistula are numerous. They include iridectomy, electrolysis, cauterization of the diseased area, covering it with a conjunctival flap, excision, bruising of the walls of the fistula with forceps, and the introduction of a suture.

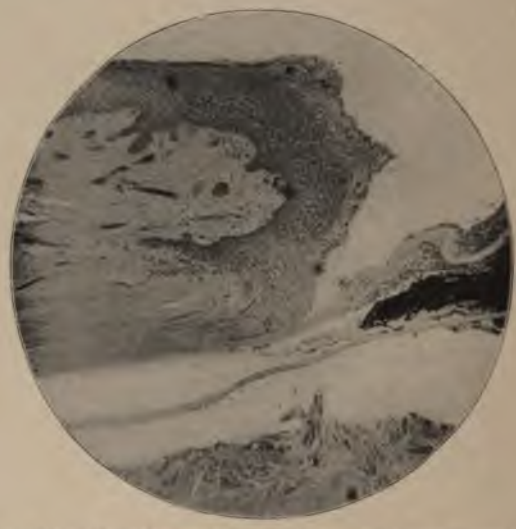


Fig. 243.—True corneal fistula, showing downgrowth of epithelium. (AUTHOR.)

(Photomicrograph by DR. H. P. WELLS of a section lent by DR. W. E. FISCHER.)

Iridectomy will save some cases of fistula. It acts advantageously in two ways: (1) by reducing intra-ocular tension and (2) by removing a tag of incarcerated iris-tissue which prevents healing. To be of value in the latter instance it must be performed early, since iris-tissue which is incarcerated in the lips of the corneal wound soon undergoes changes, becoming intimately united with fibrin and exudates.

Cauterization, theoretically, should be the ideal operation. It certainly is a rational procedure to destroy the epithelium which has downgrown into the cornea and prevents healing. In the case of a spurious fistula—*i.e.*, a small bulging scar—it is equally rational to use the cautery. But, unfortunately, by the methods heretofore in use, a thorough cauterization of the fistulous area cannot be accomplished without injury to the

crystalline lens. Thus, the surgeon is likely to produce a cataract by attempting to cure a fistula. The depth of the normal anterior chamber is only 2.6 millimetres and, as stated above, the chamber is temporarily abolished whenever a corneal fistula is open. To obviate the dangers, and at the same time to obtain the benefits of cauterization, the author has devised the operation which is described in the latter part of this chapter. Excision and suturing have been proposed. They must be regarded as dangerous and unnecessary procedures. Many years ago de Wecker, believing fistula to be caused by the eversion of the membrane of Descemet, devised the following operation: A fine straight forceps was introduced. The wall of the fistulous tract was seized and torn so as to denude the true corneal tissue. A solution of atropin was used and a compress bandage was applied, being removed and reapplied daily for two weeks. In this way de Wecker cured a case which for ten months had resisted other methods of treatment. Heymann states that in some cases a permanent cure has followed the covering over of the fistulous area with a conjunctival flap. It is assumed that this procedure is preceded by a bruising of the walls of the canal and a removal of the tag of incarcerated iris.

Electrolysis has been employed by Cornwall, who cured a case of corneal fistula in which an unfavorable prognosis had been given. The opening was situated near the corneal periphery and was so small that nearly normal tension was maintained. His procedure was as follows: The point of a jeweler's broach was bent at a right angle to the main shaft, the bent portion measuring about one millimetre in length. Under cocain anesthesia this was inserted into the fistula. The dispersing electrode was placed on the cheek. A current of the strength of $^1/_4$ milliampère was used. The bent portion of the electrode was rotated so as to describe a circle, and in this way the cornea and iris were eroded. The eye was bandaged for two days. A complete cure followed.

OPACITIES OF THE CORNEA.

In this place will be considered those opacities of the cornea which are stationary, reference having been made elsewhere in this chapter to the various changeable opacities which accompany keratitis. A stationary opacity, which is usually permanent, may be of inflammatory or of non-inflammatory origin. A typical example of the latter is the arcus senilis.

Arcus Senilis (Gerontoxon Corneæ).—This is an opaque circle situated within the corneoscleral margin, from which it is separated by a narrow strip of transparent cornea. It is due to hyalin degeneration, and is found chiefly in middle-aged and elderly subjects, although it has been seen in children. It is hereditary in some families. It appears first as a gray, semilunar arc in the upper part of the cornea; this is followed by a similar arc below, and finally the arcs unite to form a ring. The formation of arcus senilis must be regarded as physiologic in old people. The condition

does not call for treatment and wounds through this part of the cornea—for example, the incision for cataract extraction—heal kindly.

Opacities due to Keratitis.—A faint opacity of the cornea which is visible only to careful (oblique) examination is known as a *nebula;* an opacity which appears grayish by daylight is called a *macula;* and an opacity which causes disfigurement by its whiteness and density is named *leucoma.* If an adhesion of the iris to the cornea exists, the last-named opacity is called an *adherent leucoma.* All of these opacities reduce vision, and some cause positive disfigurement.

The amount of reduction in visual acuity will depend on the density of the opacity, its location with reference to the pupil, and the amount of distortion of the corneal surface. In these cases vision for distance is much more reduced than is near vision. The opacity produces an irregular astigmatism, which often does not admit of improvement under glasses or the use of a stenopaic slit; and, furthermore, it is a fruitful source of nystagmus, strabismus, myopia, and amblyopia. An adherent leucoma may produce secondary glaucoma. Small corneal cicatrices sometimes cause keratalgia, which is evidenced by lacrimation, pericorneal injection, and ciliary pain due to strangulation of nerve-filaments. Corneal scars may undergo hyalin, fatty, or keloid degeneration. Often the hyalin substance becomes impregnated with lime salts, as can be demonstrated microscopically. Incrustations of metallic salts are visible to the naked eye, and often follow the use of solutions of the acetate of lead in ulcerative keratitis. The result is a white scar. As has been said elsewhere, the salts of lead should never be used in the treatment of ulcer of the cornea. In some instances it seems impossible to demonstrate the presence of lead in corneal scrapings, but recently Ellett has found the usual lead reactions in microchemic examination.

Treatment. — Many remedies have been tried in the treatment of corneal opacities. Mercurial salves have been used, followed by massage of the eye through the lids. Calomel and other powders have been dusted on to the cornea, lotions have been sprayed on to the cornea, and jequirity has been brushed upon the conjunctiva, all with the purpose in view of causing irritation, stimulation, and absorption of the scar-tissue. Electricity has its advocates. Alleman favors the galvanic current; the cathode in the form of a silver cup, seven millimetres in diameter, in which a drop of mercury is placed, is applied to the cornea, while the anode of sponge is placed on the cheek. At first the *séances* last one minute and from $^1/_4$ to $1\,^1/_2$ milliampères are used; later they last four or five minutes, and 3 or 4 milliampères are used. It is said that the greatest improvement obtains in the scar-tissue following interstitial keratitis, although opacities following corneal ulcers are also benefited. The treatment is given under holocain or cocain anesthesia, and is repeated every third or fourth day.

Massage of the cornea may be used indirectly—*i.e.,* through the eyelid; or directly—*i.e.,* by applying pressure with a lens-spoon or spatula

against the opaque spot. Indirect massage with powdered boric acid is sometimes followed by improvement. Calomel insufflations should not be used in the eyes of persons who are taking the iodids, since the iodid excreted in the tears forms with calomel an irritant compound. In case massage or insufflation is used, or both together, benefit may follow the application of hot packs.

The administration of 2- or 3-grain capsules of thiosinamin, continued for two or three months, has sometimes resulted in the disappearance of nebulæ. Some cases of dense corneal opacity have shown marked improvement under its use (Suker).

When such measures have been faithfully tried and seem to have lost their value, nothing remains but surgical treatment. The measures of this class are numerous, but attention will be directed chiefly to three: iridectomy, surface-needling of the cornea, and keratoplasty. In case of adherent leucoma a certain amount of clearing of the opacity follows the division of adhesions of the iris to the cornea by means of von Graefe's knife or by an iridectomy. However, vision may be made worse by iridectomy because of admission of diffuse light. Tattooage of the cornea may be resorted to under such circumstances. In case of great reduction of vision from a corneal scar, an optical iridectomy may give a brilliant result.

Seeley and de Beck have observed marked improvement in vision in eyes which had been tattooed and in which the pigment had disappeared. This observation lead them to adopt the usual steps of tattooing, omitting the India ink. This surface-needling of the cornea is simply pricking the opaque tissue with the needle. The conjunctiva is washed with sterile water and the pricking is done with a sterile instrument. The operation may be repeated at intervals of about a year. In many cases vision will be increased from the counting of fingers at seven feet to V. $= {}^{20}/_{200}$ or even ${}^{20}/_{50}$.

Opacities due to a deposit of lead in the cornea are to be treated by abrasion. Keratoplasty may be of value in cases of total leucoma in which perception of light is present.

INJURIES OF THE CORNEA (TRAUMATIC KERATITIS).

Its exposed position renders the cornea liable to injuries of many kinds. These include lacerated, incised, and punctured wounds; pressure-injury during instrumental delivery; burns and scalds; frostbite from excessive application of cold, etc.

Injuries from Heat and Chemicals.—In this class may be placed those cases in which the cornea is injured by heat and chemicals. Hot water, burning gases, hot oil, lard; the ends of matches, cigars, or cigarettes; and curling-irons are agents often causing injury to the cornea. Naturally the interpalpebral part of the cornea is most violently affected by these agents.

SYMPTOMS.—After injury the eye will be painful; there will be profuse lacrimation, photophobia, pericorneal and conjunctival injection, with more or less swelling of the conjunctiva and eyelids. There is a mucopurulent or purulent discharge. The corneal lesion may be limited to the epithelial layer, producing a thin, whitish film, which is soon regenerated; or the deeper layers may be involved, producing a dense, opaque scar or causing perforation. In such severe injuries of the cornea it is the rule that the conjunctiva likewise is seriously involved (Fig. 1, Plate X), leading to adhesions between the globe and eyelids.

TREATMENT.—If the injury is caused by an acid, and the case is seen early, an attempt at neutralization can be made with a solution of bicarbonate of potash (saleratus). If lime, caustic potash, or other alkali has produced the injury, it may be neutralized with dilute vinegar or lemon-juice. The foreign substance should be removed and the conjunctiva should be washed thoroughly with sterile water or with physiologic salt solution. Pain is kept under control by the application of cold compresses, which are to be frequently changed, and by the instillation of a 1-per-cent. strength solution of holocain. Denuded areas of conjunctiva may be kept from growing together by frequently drawing the lid away from the globe; however, if the fornix is involved, a symblepharon will surely form. In case both ocular and palpebral conjunctiva are burned, and the involved ocular area is small, sutures may be used to close the gap in the ocular conjunctiva and adhesions can thus be prevented. Where two raw surfaces are in contact the daily use of the probe and the application of an unirritating ointment may prevent or limit adhesions.

Mechanical Injuries of the Cornea.—These injuries may be inflicted in any one of many ways, and may be complicated by the lodgment of foreign bodies. Compression injuries are sometimes met with in the newborn as the result of instrumental delivery. In such cases there will be bruising of the eyelids and conjunctiva, with dulling of the anterior corneal layers, followed by an obliquely placed scar. In the course of several months such opacities may entirely disappear. Scratches may be caused by the finger-nail, a twig, etc., and are extremely painful and liable to infection. Such injuries may be followed by recurrent bullous keratitis. Not rarely the attacks recur several times a year, for two or three years. In this disease the attacks invariably come on in the morning, the patient awakening with pain, pericorneal injection, and lacrimation. Punctured and incised wounds of the cornea usually involve other structures (the sclera or iris and lens). Such wounds, without the lodgment of a foreign body, may come from the unskillful use of a needle or from the impact of a thorn. Contusions of the cornea are not uncommon, and generally other parts of the eye are injured at the same time. Injuries by flying pieces of coal are often followed by corneal necrosis.

SYMPTOMS.—Injuries to the cornea are generally followed by severe pain, photophobia, lacrimation, pericorneal injection, and reduction in

vision. There is a scratching sensation, leading the patient to believe that a foreign body is present. The injured eye should be examined by oblique illumination. Denuded areas can be outlined accurately by the use of fluorescin solution.

PROGNOSIS.—Corneal injuries are peculiarly liable to be followed by infection. This is particularly the case in the presence of dacryocystitis. Deep wounds always leave a scar, which, if located over the pupil, interferes with vision. Penetrating wounds, followed by loss of the aqueous humor and sometimes by prolapse of the iris, are chiefly of interest in connection with the damage done to the iris, lens, and deeper structures of the eye.

TREATMENT.—Patients with corneal injuries are clamorous for relief, and holocain should be used to relieve pain. The conjunctiva should then be flushed with a weak bichlorid solution (1 to 5000), atropin should be instilled, and the eye should be covered with a light gauze dressing. If the injury has been infected, the treatment will be the same as outlined above, with the addition of the destruction of the infected area by the curette or galvanocautery.

Penetrating wounds of the cornea require careful treatment lest infection occur and spread to the deep ocular structures. The cornea and conjunctiva should be washed with a sterile bichlorid (1 to 5000) or salt solution (0.5-per-cent. strength). If a prolapsed iris is present, it should be cut off. To prevent secondary infection, the wound should be covered with a conjunctival flap. If the wound is situated at the periphery of the cornea, the simplest method of covering it will be to loosen the conjunctiva all around the cornea, and insert a purse-string suture. This is to be tied and is left *in situ* for several days. If the wound involves the centre of the cornea, a loosening of the conjunctiva with the excision of a suitable area of this membrane, will be required. In simple, clean-cut corneal wounds this method of treatment may not be necessary. In lacerated wounds it should be adopted.

Healing of Corneal Wounds.—Since pathologists have made it the battle-ground on which to test their theories, much has been written concerning the healing of wounds of the cornea. Systematic writers distinguish three stages in the reparative process:—

1. The stage of simple adhesion.

2. The stage of temporary closure of the wound through epithelial plugging.

3. The stage of definite scar-formation.

It is impossible here to discuss the minute changes which occur in the healing process. For such information the reader is referred to systematic treatises on surgical pathology.

Hemorrhage into the Cornea.—A very rare accident is hemorrhage into the layers of the cornea. As a result of trauma, blood is effused between the layer of Descemet and the substantia propria, and slowly spreads until it obscures nearly the whole iris. After a period of two or three

weeks the blood is absorbed, leaving some pigment stippling the cornea. In a case of hemorrhage into the cornea the diagnosis must be made by oblique illumination, and it will be necessary to exclude hemorrhage into the anterior chamber. Fig. 244 shows a hemorrhage into the cornea as it appeared one day after the eye had been struck by a flying cinder, and in Fig. 245 the same eye is shown as it appeared one day later.

Foreign Bodies in the Cornea.—The lodgment of a foreign body in the cornea is a common accident. Pieces of iron, steel, brass, copper, coal, stone, or wood are among the common missiles. A foreign body may injure only the epithelium or it may penetrate deeply into the cornea. The seriousness of such an accident depends somewhat on the depth to which it penetrates, but chiefly on the condition of the foreign body. If aseptic, little harm results, aside from the formation of a scar when penetration has been deep; if septic, it may set up a rapidly destructive necrotic process which often ends in iritis, perforation of the cornea with its attendant evils, or panophthalmitis. The size of these missiles varies greatly. Most of them are

Fig. 244.—Hemorrhage into the cornea. (After DE BECK.)

Fig. 245.—The same eye one day later. (After DE BECK.)

minute pieces which may defy recognition until after the use of oblique illumination, with or without the aid of fluorescin solution. Pain, photophobia, lacrimation, and pericorneal injection follow the injury.

TREATMENT.—The indications for treatment are: (1) removal of the foreign body, (2) the prevention of infection, and (3) the treatment of complications. To remove a foreign body lodged in the cornea is usually such a simple affair that some practitioners, it is to be feared, do not observe the rules of asepsis. The instruments required are a speculum, fixation forceps, corneal spud, and a cataract-needle. Rarely will it be necessary to use other instruments, although it is conceivable that a small foreign body, lodged deeply in the cornea and projecting into the anterior chamber, may best be removed by passing a keratome into the chamber and pressing it against the foreign body, while a cataract-needle is applied externally to pry the substance out of its bed. In an ordinary case the surgeon proceeds as follows: The eye having been anesthetized with holocain, the speculum is placed in position and the eye is held quiet with the fixation forceps in the left hand, while the right guides the spud or cataract-needle. The in-

strument is placed under the foreign body and lifts it out. If imbedded deeply, the cataract-needle can be used to dig it out. The conjunctiva, which previous to the operation should have been flushed with a 1 to 5000 bichlorid solution, is again washed. Usually it is not advisable to bandage the eye. In case the injury is of severe character, atropin and a gauze dressing should be used. The patient should be seen once a day until he is well. If infection has occurred, the case will require atropin, intermittent applications of moist heat, and possibly the galvanocautery. In all operative procedures about the eye only sterile instruments should be used. The practice of removing cinders, etc., with toothpicks, knife-blades, and other unclean instruments is a prolific source of corneal ulceration.

If the foreign body rests against the posterior surface of the cornea, and its shape or position is such as to render its removal impossible by the procedures which have been described, the surgeon may resort to the temporary renversement of a corneal flap (Gayet's operation).

OPERATIONS ON THE CORNEA.

Abrasion of the Cornea is the scraping or cutting off of the superficial layers. It can be done with a cataract-needle, with the knife of von Graefe or that of Beer, or with a small scalpel or curette. The operation is performed in a limited area, as a necessary step in the removal of foreign bodies, and as a more extensive procedure in the removal of deposits of lead in the cornea, or in the removal of opaque masses in ribbon-shaped keratitis. Deschamps has advocated curettement of the cornea after the removal of the apex of a pterygium. Abrasion (curettement) is frequently done for the cure of infected corneal ulcers.

Paracentesis of the Cornea.—This operation can be performed with a cataract knife or needle or with a small keratome. The other instruments required are a speculum and fixation forceps. The puncture can be made in any portion of the periphery of the cornea. A few drops of aqueous humor are permitted to escape, this being facilitated by a turning of the instrument on its axis. The operation is indicated by an increase of intraocular tension: glaucoma, hydrophthalmos, iritis. Paracentesis in glaucoma is especially valuable in the glaucoma following cataract extraction. After the evacuation of aqueous a miotic is to be used. Some of these cases will recover without further treatment; often, however, an iridectomy is more efficient. If paracentesis is indicated in corneal ulcer, the opening should be made not through the floor of the ulcer, as is usually advised, because of the danger of infecting the eye, but, with care, a paracentesis can usually be made through normal tissue, and infection should not follow.

Cauterization of the Cornea, which is useful in the treatment of infected cases and in conic cornea, can be done by chemic, thermic, or electric means. The galvanocautery is preferred. After using atropin if the ulcer is central, or eserin if it is peripheral, a local or general anesthetic is em-

ployed. The galvanocautery is brought to red heat and is applied to the ulcer. After all the sloughing material has been destroyed, atropin is used and the eye is bandaged.

In the treatment of conic cornea the reaction and scar are proportionate to the extent and intensity of the cauterization. Moderate cauterization is followed by prompt healing and limited scars, while extensive cauterization causes iritis, suppuration, and sloughing of the cornea. Perforation of the cornea should be avoided, but is not necessarily followed by bad symptoms. Knapp uses a convex, disc-like electrode at a dull-red heat, and superficially burns an area from the centre of the cornea down and outward about four millimetres in diameter. After a few moments a smaller zone within the former is treated the same way, and, last, the cold electrode is placed in the centre of the area and withdrawn immediately after the platinum becomes red. Holocain is the anesthetic used. In the treatment of conic cornea by cauterization at least one-half of the pupillary area should be spared.

Excision of the Cornea (Staphylectomy).—This operation may concern the partial or total removal of the cornea. Partial removal can be

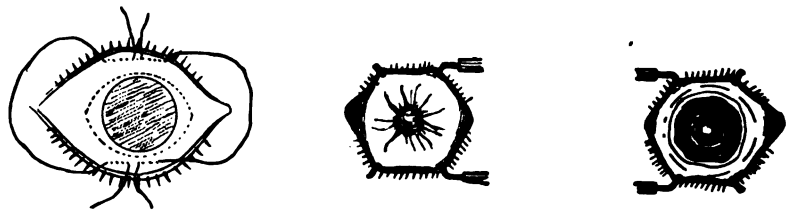


Fig. 216.—Operation for corneal staphyloma. (KNAPP.)

effected by means of a cataract-knife or by a trepan, and has been performed for keratoconus, staphyloma, corneal fistula, and large central opacities. Total excision is done for complete staphyloma and for various conditions as a step of Mules's operation.

As regards partial excision, Berry's method of treatment of partial staphyloma is simple and effective. A cataract-needle is passed through the base of the protrusion and a cataract-knife is used to cut away the desired amount of cicatricial tissue.

Total excision of the cornea may be done for total staphyloma where there is a complete loss of vision. Beer treated these cases by making an incision below with the triangular knife which bears his name, and completed the excision with scissors. The wound gradually closes with a white cicatrix. Critchett's operation is made by first passing four or five long curved needles, armed with silk thread, through the anterior part of the globe. The staphyloma is then abscissed, the needles are drawn through, and the threads are tied. Suppuration in the operated eye, or sympathetic ophthalmitis, may occur after this procedure. Knapp, to avoid the danger of passing needles and threads through the ciliary body, devised the

following operation: A curved needle is passed through the conjunctiva and outer scleral layer at a point four millimetres behind the cornea, above and below (Fig. 246), both nasally and temporally. The threads are left in position, the staphyloma is abscissed according to Beer's method, the lens is removed, and the threads are tied. By this operation the wound is closed by four vertical threads, each loop forming a suture. When they are tied, the wound closes like the mouth of a purse. De Wecker, after excising the staphyloma, covers the defect with conjunctiva drawn by a purse-string suture. Other methods of corneal excision have been devised by Carter, Panas, and Czermak.

The accidents following these operations are immediate and remote. Among the former are hemorrhage and suppuration. Intra-ocular hemorrhage, when occurring during the operation, results in expulsion of the vitreous humor, while the retina and chorioid appear in the wound. This accident renders it necessary to change the operation into an evisceration, with or without the insertion of an artificial vitreous body, as in the judgment of the operator seems best. Suppuration, which was frequent before the era of asepsis, and which occurred in case sutures were not used to close the wound, is now a rare accident. The remote accidents are glaucoma, plastic chorioiditis, the formation of exuberant granulations, and sympathetic ophthalmitis. Glaucoma does not occur unless the lens is left *in situ* or a large part of the staphyloma remains. If tardy suppuration or plastic chorioiditis occurs, the stump must be curetted. Polypoid growths in the stump call for the use of the cautery. Sympathetic ophthalmitis, so terrible a complication, is fortunately of rare occurrence. If threatened, the stump should be enucleated and the optic nerve resected to the optic foramen. The results of excision of the cornea are very beautiful. The stump serves to support an artificial eye, and the cosmetic effect is all that could be expected.

Keratotomy.—This operation, the incising of the cornea, is a step in the cataract operation, in iridectomy, in the removal of foreign bodies from the anterior chamber, etc. It is not to these that reference is here made, but to the keratotomy of Saemisch, which is applied to the treatment of ulcus serpens. This kind of ulcer often fails to improve under treatment less heroic than section of the cornea.

Saemisch's Incision.—This operation is indicated in rapidly spreading (serpiginous) ulcers of the cornea, which have involved a considerable area and have resisted ordinary therapeutic measures. The necessary instruments are a speculum, fixation forceps, and a narrow von Graefe knife. A spatula, iris forceps, and scissors should be provided as reserve instruments.

A local, or preferably a general, anesthetic should be used. The speculum having been introduced and the fixation forceps applied, the surgeon punctures the cornea in its horizontal diameter, at a point one millimetre from outside the ulcer. The counter-puncture is made at a corresponding

point. The section is then completed by gentle sawing movements, care being taken not to turn the knife on its axis lest the iris become prolapsed. The completion of the section is followed by a flow of aqueous humor, which carries much of the hypopyon with it. The fibrinous mass which engages the wound is now to be removed by means of the forceps. A gauze dressing and bandage are to be applied, and should be changed several times a day. Some authorities advise that the wound be reopened by the spatula, this procedure being repeated daily for five or six days. Such treatment is likely to be followed by intra-ocular infection. Hence it is unwise. A collyrium (made with boric acid, sublimate solution, or argyrol) should be used daily.

Among the complications are: prolapse of the iris, which, since it cannot be excised, results in anterior synechia and later may cause secondary glaucoma; loss of the lens and vitreous body, which will be more likely to occur under local than under general anesthesia, often leads to panophthalmitis. Since Saemisch's incision is employed only in desperate cases, the complications are of little importance.

Operation for Fistula of the Cornea.—To obviate the danger of injury to the lens during the treatment of a corneal fistula the author proposes the following operation: A keratome is to be introduced into the anterior chamber as in the first step in the operation of iridectomy. The tip of the instrument is to pass beyond the fistulous area, and the instrument is then to be held *in situ* while a cautery is used to destroy the epithelial lining, the tag of iris-tissue which is often present in the fistula, and the bulging bleb of Descemet's membrane. The keratome is to be withdrawn carefully to avoid injury to the lens. Atropin and a compress bandage are to be used in the after-treatment. During the operation the lids should be separated by a speculum, and the eyeball should be fixed with forceps. It is necessary that the forceps should be placed several millimetres behind the point at which the keratome is to be introduced. If the forceps be placed on the globe at a point opposite the place at which the keratome is to be passed, the eye may be subjected to sufficient pressure to cause a rupture of the weakened area. The operation must then be postponed until such time as the anterior chamber shall have re-formed.

Temporary Renversement of a Corneal Flap (Gayet's Operation) is performed for the removal of foreign bodies which rest against the posterior surface of the cornea and cannot be extracted by ordinary means. Under general anesthesia and scrupulous cleanliness, the following operation is done: 1. With a von Graefe knife the surgeon makes a corneal flap, which is somewhat similar to that of a cataract operation. The size and position of the flap must be regulated by the site and dimensions of the foreign body. 2. The flap is seized with forceps and is reversed,—*i.e.*, is bent upon itself—thus permitting the removal of the foreign body. 3. The flap is replaced. A suture can be used if deemed necessary. Atropin and a compress bandage are used in the after-treatment.

Tattooing of the Cornea.—This ancient operation was revived in 1869 by de Wecker and is employed chiefly in those dense opacities which form a marked impediment to vision and constitute a noticeable blemish. The operation should not be done upon thin and staphylomatous corneæ, since it may be followed by iritis, iridocyclitis, or glaucoma. According to Noyes, the best results follow where the cornea is of normal thickness or is abnormally thick. In any event, tattooing is not to be resorted to until the pathologic process is quiescent. Colored tattooage was employed by the ancients, but at present only black is used. The operation requires a quiet (non-irritable) eye, a patent drainage apparatus, and an aseptic conjunctiva. Most text-books advise that the white spot be made black, the Taylor needle being the instrument used. While this method undoubtedly improves its appearance, the eye looks unnatural, because its most characteristic feature—a black central pupil—is wanting. To obtain this, Barck advises the surgeon to tattoo a round central spot, leaving a surrounding area unstained, so that the pupil will show by contrast, while the periphery is tattooed and made to resemble as far as possible the iris of the normal eye. The making of a round pupil is much simplified by employing Barck's instrument (Fig. 247) consisting of two parallel cutting rings, the inner

Fig. 247.—Tattooing instrument. (Barck.)

marking the limits of the pupillary space, while the outer marks the inner border of the iris-ring. These boundaries having been defined, the tattooing is finished by means of an instrument consisting of a bundle of needles. Under local anesthesia, the eyeball being held with blunt forceps to avoid tearing of the conjunctiva and staining of this membrane, a number of obliquely directed stabs are made through the epithelial layer, and the ink is rubbed into the openings. The eye is irrigated with sterile water and the effect noted. The process is repeated at intervals of two or three weeks until the proper coloration has been secured. Atropin is instilled and a bandage is applied for a few days. The substance used is the best quality of Chinese or India ink, made into a sterile solution as thick as oil or paste. The result may remain for many years, but in some cases the pigment is absorbed in a few months.

Among the rare accidents following this operation Terson mentions infection of the cornea (causing panophthalmitis), perforation with the tattooing needle, iridocyclitis, and sympathetic ophthalmitis. Recently Trousseau has observed a case where tattooing of an eye with adherent leucoma produced iridocyclitis and sympathetic ophthalmitis, with resulting blindness. The same author has once observed sympathetic ophthalmitis following tattooing of the cornea for non-adherent leucoma.

During the operation care should be taken not to tear the conjunctiva with the fixation forceps, since this will result in tattooage of the conjunctiva. To avoid this accident it has been proposed to use fixation forceps made of bone, ivory, or rubber.

After the operation, the particles of charcoal are held in the superficial corneal lamellæ beneath the epithelium. The black coloration is not permanent. If the patient is seen several years after the tattooage the grains of charcoal will be found to have moved, occupying an excentric position or even invading the transparent tissue.

Keratoplasty.—In cases of total leucoma with good perception of light, the transplantation of a piece of the cornea of a lower animal into a bed in the eye of a human being has been practiced. The operation dates from the time of Reisinger (1828), and has claimed the attention of many able surgeons. Von Hippel, who has devised a trephine for the rapid and accurate execution of the operation, has met with some encouraging results, some of his patients having been able to see through the transplanted tissue for many months. He uses a button from a rabbit's cornea for transplantation. The insertion of an artificial cornea, or rather the insertion of a piece of glass into the cornea, was practiced by Nussbaum (1856). While immediately successful, the end was a failure.

In recent years Fuchs, who possesses an abundance of clinical material, has often successfully transplanted a piece of the human cornea, using for this purpose the immediately enucleated eye of another patient. This, of course, can be done only under favorable surroundings, in large clinics, and under strict adherence to rules of asepsis. The eye from which the disc is to be taken, and that into which it is to be inserted, must be made surgically clean. Two surgeons then work simultaneously. While the one is removing a disc from the leucomatous eye the other is enucleating the eye of the patient who furnishes the graftive material. It is necessary to avoid opening Descemet's membrane. If this occurs, aqueous humor will come in contact with the graft and the result will be a failure.

CHAPTER IX.

DISEASES OF THE SCLERA.

CONGENITAL ANOMALIES.

THESE include melanosis, tumors, and certain staphylomas. *Congenital pigmentation* of the sclera (melanosis scleræ) presents either small spots or diffuse areas of discoloration. Generally there are similar conditions in the iris and chorioid. It is around the openings for the passage of the anterior ciliary veins and about the optic-nerve entrance that branching pigment-cells are found. Acquired pigmentation occurs in some cases of Addison's disease.

TUMORS OF THE SCLERA.

Tumors of the sclera are of rare occurrence. Serous cysts have been recorded by Rogman and Hasner. The origin of such tumors is in doubt. Some authorities hold that they are dilations of the canal of Schlemm. Others state that they are due to a congenital fistula from the anterior chamber into the sclerotic, which permits the aqueous humor to pass between the lamellæ of the sclera. Some authors attribute them to encysted chorioidal exudates producing small staphylomata of the sclera. The diagnosis may present difficulties. In Rogman's case the tumor collapsed on puncture without change in the anterior chamber. Among the solid tumors a fibroma was described by Saemisch and an osteoma by Watson. Malherbe reported a tuberculous tumor of the sclera. Quaglino described a case of telangiectasis of the sclera. Of 137 malignant tumors of the exterior of the eyeball, Noyes found that 20 arose from the sclera and 31 from the corneoscleral junction. The proper treatment of scleral tumors is excision.

INFLAMMATIONS OF THE SCLERA.

Affections of the sclera belong to the class of uncommon ophthalmic diseases. The sclera is slow to take on inflammation, and, when once scleritis is inaugurated, recovery is slow, as might be expected from such a structure. Scleritis may be superficial or deep, acute or chronic, diffuse or circumscribed. Of the inflammations affecting this part, the most common is that known as episcleritis, in which the pathologic process involves the loose tissue over the sclera and possibly the superficial scleral layers also.

Episcleritis (Superficial Scleritis) is a localized inflammation which produces an exudate into the episcleral tissue. The exudation causes a protuberance, which is usually rounded or flat and is situated at a distance

of several millimetres from the cornea. The mass is attached to the sclera, and the conjunctiva can be moved over it. Two kinds of injection are visible in a case of episcleritis: a superficial hyperemia of the conjunctiva and a deeply placed violet-colored injection from the episcleral vessels (Fig. 6, Plate X). The eye is red only in the neighborhood of the nodule, which is hard and, if it enmeshes one of the ciliary nerves, is acutely sensitive to the touch. The subjective symptoms in episcleritis may be mild or severe, but the course of the disease is subacute or chronic. In some cases photophobia, lacrimation, and a dull, heavy pain are symptoms. After several weeks the affection usually disappears, the nodule is absorbed, and either no trace is left or a slate-colored patch remains to mark the site. In some cases other nodules form until the entire circumcorneal zone is involved. A characteristic feature of the disease is the tendency to recurrence, either in the old site or elsewhere. The process may persist for months or even years. The inflamed patch frequently resembles phlyctenular conjunctivitis, with which it may be confounded. Although, as a rule, the cornea is unaffected in episcleritis, if the nodule is located near the cornea, an infiltration of the latter may occur during the height of the disease. Iritis is rarely a complication. Both eyes may be involved in episcleritis.

Etiology.—The etiology of episcleritis is somewhat obscure, although it undoubtedly can be attributed to gout or rheumatism in some cases. Exposure to cold, menstrual derangement, and scrofula are supposed causes. Syphilis is rarely an etiologic factor.

Pathology.—In episcleritis the conjunctiva, the episcleral tissue, and the superficial scleral layers are involved. The conjunctiva is hyperemic and the episcleral tissue is edematous. The superficial scleral layers show abundant fibrinous and cellular infiltration, with dilation of lymph-vessels and blood-vessels. The walls of the vessels are generally thinner than normal and are surrounded by an area of cellular infiltration. Hemorrhages may be present or absent. The inflammatory products do not tend to disintegration; they disappear by resorption. There is not a clearly defined line of demarcation between episcleritis and scleritis, transition forms existing.

Diagnosis.—In fully developed cases there can be little difficulty in the diagnosis. Episcleritis may be mistaken for phlyctenular conjunctivitis. In the latter affection there is present a denuded area of a whitish-yellow color, and the whole inflamed patch is movable with the conjunctiva.

Darier states that the diagnosis of episcleritis from conjunctivitis is facilitated by the use of adrenalin, which produces marked anemia of all the conjunctival tissue, but leaves a hyperemic spot at the level of the episcleral inflammation.

Prognosis.—Episcleritis generally lasts one or two months, but may continue longer. Recurrences are not infrequent. In general, the prognosis is favorable; exceptionally the deeper layers will be involved, leading to ectasiæ.

TREATMENT.—In the treatment of episcleritis it is necessary to attend to the general health. Gastric and uterine disorders should receive appropriate treatment. Salicylate of sodium, aspirin, and colchicum are valuable remedies for internal use. Pilocarpin by the mouth or hypodermically will often produce an amelioration of the symptoms. Massage, heat applied by the Japanese hot box, and the subconjunctival injection of a solution of bichlorid of mercury (1 to 3000) are valuable therapeutic measures. Adrenalin, applied three or four times daily, is of value. After each application gentle and prolonged rotary massage with mercurial lanolin is to be practiced (Darier). Injections of salicylate of sodium (2 per cent.), of cinnamate of sodium (hetol), or of salt solution have been recommended. As-

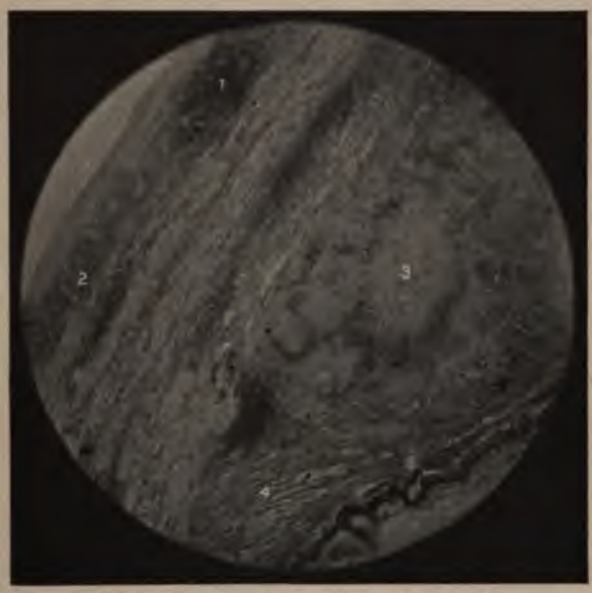


Fig. 218.—Deep scleritis. (AUTHOR.)

(Photomicrograph by DR. H. P. WELLS.)

1, 2, Superficial infiltration. *3*, Area of deep infiltration. *4*, Ciliary muscle.

tringent metals and caustics should not be used. If the cornea is involved, atropin should be applied, but with due caution in elderly persons. In rebellious cases, and in those which are prone to recur, it will be advisable to excise the inflamed patch. Scarification and curettage have been employed by some surgeons. Attention should be given to the state of the refraction and of the muscle-balance.

Fugacious Episcleritis (Episcleritis Partialis Fugax; "Hot Eye") is a form of sudden transient hyperemia of the episcleral tissue and overlying conjunctiva. It lasts for a few days and reappears at intervals varying from a few weeks to several months. There is pain, photophobia, and lacrimation, but vision is unaffected. The disease occurs chiefly in adults of a rheumatic or gouty diathesis, but children are not exempt. The

affection is supposed to be identical with that described in 1892 by Dr. Burnett, of Washington, as "a vasomotor dilation of the vessels."

The name "hot eye" was given to the disease by Hutchinson. The treatment is the same as that outlined above for episcleritis.

Scleritis (Deep Scleritis) is a much less frequent disease than episcleritis. It is a much more obscure disease, and in the early stages can be recognized with difficulty, since, in the absence of a microscopic section, it is impossible to say how deeply an episcleral inflammation has extended. Early in its history the presence of scleritis is to be inferred from the existence of complications, such as iritis, chorioiditis, and sclerosing keratitis. Scleritis may be acute or chronic, diffuse or circumscribed, anterior or posterior. As an example of the acute type, mention may be made of the inflammatory thickening of the sclera which occurs in orbital cellulitis and panophthalmitis (Fig. 291). The posterior scleritis which is found in myopia involves the chorioid, and is called posterior sclerochorioiditis (see chapter on the chorioid). As a result of inflammation, the sclera becomes softened. The normal or increased intra-ocular tension causes the membrane to bulge (ectasia of the sclera). Scleritis tends to extend until the entire cornea has been circumscribed (Fig. 249).

Early in its history scleritis presents a diffuse or circumscribed area of bluish-red or violaceous injection, situated in the ciliary region. Pain and lacrimation are prominent symptoms. The inflamed tissue resembles the patch of episcleritis, but is less sharply defined. When the acute process has subsided, the circumcorneal zone will appear of a violet color, resembling porcelain; if the sclera is much thinned, the affected area will be of a bluish tint, which is given to it by the subjacent chorioid and ciliary body. Deep scleritis generally involves both eyes, and presents important complications (iritis, cyclitis, keratitis) which seriously impair vision.

Sclerosing Keratitis.—In this affection, which is an accompanying symptom of scleritis, the corneal limbus is invaded by a dense opacity which creeps over it. Apparently the sclera has pushed its way into the cornea. The opacity may appear as a semicircle or circle, but frequently it is an irregular patch with prolongations. Its base is directed toward the inflamed sclera. The disease may be unilateral or bilateral. Iritis or cyclitis may be present or absent. The corneal opacity, which at first is gray or yellowish, may be thick and permanent, or it may be partly clear. Early in the disease tension may be increased; later on, when cyclitis, hyalitis, hemorrhages, and vitreous opacities ensue, the tension will be subnormal. Like the scleritis which causes it, sclerosing keratitis may often recur. In some cases the entire cornea, with the exception of a small pupillary area, becomes opaque.

Sclerokerato-iritis is the term applied to a complicated disease affecting the structures at the corneoscleral junction. It also is known as scrofulous scleritis, anterior uveitis, or anterior chorioiditis. Relapses are common and the disease often is intractable. It begins with the clinical signs

an anterior scleritis; soon the cornea is affected and may go on to ulceration; the iris is involved and posterior synechiæ form. Under appropriate treatment the condition improves, but relapses often occur. Such cases often end in partial or total loss of vision from cyclitis and hyalitis.

Etiology.—Deep scleritis may be due to syphilis, scrofula, rheumatism, gout, or tuberculosis. It may result from exposure to cold. Gonorrhea, when associated with synovitis, is a cause. Disorders of menstruation are often present in females with deep scleritis. Sclerosing keratitis is sometimes found in adults who apparently are of robust constitution.

Fig. 249.—Sclerosing keratitis. (After Demours.)
(Drawn by Dr. R. W. Mills.)

Pathology.—In the few unquestioned cases of scleritis which have been subjected to microscopic examination, a variety of changes has been found. In Baumgarten's case the sclera was thickened, measuring four millimetres, a condition which was due partly to an infiltration of small, round cells between the scleral bundles, partly to an increase in the number of fibres. In a case of old uveitis and scleritis Kostenitsch found abundant round-cell infiltration, numerous polynuclear leucocytes, and small hemorrhages in the sclera, and an increase in the number of scleral cells and of blood-vessels. Edema and discoloration of the scleral fibres was noted by Schirmer. As a result of degenerative changes the sclera loses its resisting power. Under such circumstances the intra-ocular pressure may lead to

the formation of ectasiæ. Atrophy does not always follow scleritis. The inflammation may result in proliferation, a formation of new tissue, and thickening of the membrane, with a formation of new blood-vessels. Since these changes are limited to that part of the sclera covering the ciliary body, and reach their greatest intensity at the points where the ciliary vessels pierce the sclerotic, it is reasonable to agree with Greeff, that deep scleritis is a secondary process, a consequence of a circumscribed uveitis which has extended along the sheaths of the vessels in the sclera. Gelatinous infiltration, purulent inflammation, and ulceration of the sclera have been recorded.

TREATMENT.—The treatment of deep scleritis includes the correction of any departure from health. In rheumatic cases the administration of salicylate of sodium, or aspirin, colchicum, salol, the alkalies, iodid of potassium, or of caffein, and the free use of water internally, will be appropriate measures. In scrofulous subjects a course of codliver-oil, with or without iodin and iron, will be in order. A change of climate will often be advisable. If tonics are indicated, iron, arsenic, and quinin should be given. In many cases diaphoresis is followed by improvement. The syphilitic cases will need mercury, which is a valuable remedy on general principles. The local use of atropin is of great importance, not only to draw the iris out of the way of harm, but to abolish accommodative effort. If the tension is increased, arecolin should replace atropin and repeated paracentesis of the anterior chamber must be resorted to, one or two drops of aqueous humor being evacuated every second or third day. The local use of moist or dry heat will be valuable to relieve pain. In some cases it will be necessary to give the patient a solution of holocain or of dionin to use whenever the pain is severe. When inflammation has subsided and has left the sclera weakened, the prolonged use of a mild miotic, combined with massage of the eye with a bland mercurial ointment, will serve to promote the absorption of the interfibrillary deposits. For the same purpose, thiosinamin (gr. ii or iij, three times a day), given in capsules, may be of value.

PROTRUSIONS OF THE SCLERA.

Ectasiæ of the Sclera (Scleral Staphylomata) are classified as anterior, posterior, and equatorial. Anterior scleral and equatorial staphylomata appear as bluish-black or grayish projections. Their color is due to the thinned sclera permitting the dark chorioid to appear. In anterior ectasiæ the limbus of the cornea forms the anterior border of the projection; in some cases both cornea and sclera participate in the bulging. Anatomic examination of enucleated eyes shows two forms of anterior scleral ectasiæ, the ciliary and the intercalary. The former shows a bulging of that part of the sclera which is lined by the ciliary body, while the latter is a protrusion between the ciliary body and the corneal margin.

Equatorial ectasiæ can be seen only when the eye is turned strongly to the side opposite the projection. Ectasiæ occur at one or more places,

where the venæ vorticosæ pass from the globe, but are said not to form a ring such as sometimes occurs in anterior staphyloma. They are the result of chronic chorioidoscleritis. On microscopic examination the chorioid and sclera are found atrophic and adherent.

The posterior ectasiæ involve the posterior segment of the globe. They cannot be seen except the eye be removed or the ophthalmoscope be used. These posterior projections are divided into (1) the posterior staphyloma of Scarpa and (2) the posterior staphyloma of von Ammon. The former is a protrusion situated at the temporal side of the optic-nerve entrance. If large, it involves the nerve itself. Arlt discovered that this form of ectasia is frequently the cause of myopia (axial myopia). The antero-posterior diameter of the globe is elongated, and the fundus shows a white crescentic patch embracing the temporal side of the disc. The posterior scleral pro-

Fig. 250.—Equatorial ectasiæ of the sclera. (After von Ammon.)

Fig. 251.—Cirsoid ciliary staphyloma. (Vossius.)

trusion of von Ammon lies below the posterior pole, and is a congenital condition which arises from incomplete closure of the fetal eye-cleft. At the present day it is best known by the name *inferior conus*. Often in such cases there is an accompanying coloboma of the chorioid and iris. The conditions described above are partial ectasiæ. It is now necessary to speak of

Total Ectasia of the Sclera.—While in the adult the sclera is rigid, and gives way only in certain weak places, in the young subject the eyeball can be enlarged in every direction. Such an ectasia of the sclera is often accompanied by an enlargement of the whole cornea (megalocornea) or by a corneal staphyloma. In this connection the reader should refer to congenital anomalies of the cornea.

The causes of scleral ectasia include those factors which either diminish the resistance of this coat or increase intra-ocular pressure. Thus, glaucoma

and exclusion of the pupil by iritis belong to the latter class, while diminished resistance of the sclera follows scleritis, tumors, gummata, tubercular nodules, injuries, and the congenital condition mentioned above.

The results of scleral ectasia are loss of vision from increase of tension, great disfigurement in the anterior and equatorial forms, and constant irritation from exposure of the protruding mass after it reaches such dimensions that the lids cannot cover it. The posterior protrusion in staphyloma often leads to great increase in the near-sight without producing increased tension. Vision is often much reduced in these cases. The ectasia of von Ammon remains stationary, and, since it is situated below the macular region, it does not impair vision.

TREATMENT.—Anterior and equatorial ectasiæ of the sclera should be treated by iridectomy, for the purpose of reducing tension. If this is accomplished, the process stops. If iridectomy cannot be performed, the eye is to be left to its fate, and ultimately an enucleation will often become neces-

Fig. 252.—Posterior staphyloma of Scarpa.

Fig. 253.—Ciliary staphyloma. (PAGENSTECHER and GENTH.)

sary. The treatment of the posterior staphyloma of Scarpa is discussed under the head of "Chorioiditis."

INJURIES OF THE SCLERA.

The sclera may be bruised, burned, or cut. Bruises result from contusions, compression, or concussion, and necessarily concern the other tunics. The sclera may be ruptured as the result of the direct or indirect application of violent force (Fig. 6, Plate XII). The line of rupture is generally placed concentrically with the cornea, at a distance of two or three millimetres from the limbus, and is frequently situated above the horizontal meridian (Praun). The length of the rupture varies from three to twelve millimetres. Wounds of the sclera may be divided into (1) *those without* and (2) *those with* the lodgment of a foreign body within the eye.

Wounds of the sclera without the lodgment of a foreign body may be incised, contused, or punctured. They may be clean cuts or lacerations, and are caused by a variety of agents, varying from a blow by the fist to cuts by knives, pieces of broken glass, birdshot, or chips of metal. They are always serious by reason of the injury done to other parts,—such as the

ciliary body, chorioid, and retina,—and because of the danger of infection. Often the vitreous protrudes from the wound, or the lens and a large part of the vitreous humor will be lost. The sclera may be ruptured by blunt instruments while the conjunctiva is intact over it, but such injuries are rare. Usually a scleral wound is easily recognized, and presents a portion of the iris, ciliary body, chorioid, or vitreous as a hernial protrusion. The vitreous will be filled with blood if the wound is situated posteriorly; if far forward, there will be hemorrhage into the anterior chamber. Small scleral wounds may be covered by a subconjunctival hemorrhage, in which case reduced tension will be a valuable diagnostic sign. Clean cuts of the sclera and some lacerated wounds, with incarcerations of part of the uveal tunic, often heal with surprisingly little reaction, and the same result may occur after loss of considerable vitreous and the lens. In favorable cases healing occurs in four or five weeks.

Rupture of Schlemm's Canal often occurs after a contusion of the limbus by a blunt body, and follows incomplete rupture of the ocular capsule. Under such circumstances, blood escapes from the canal and forms a coagulum at the periphery of the anterior chamber. The injury is followed by the appearance of diffuse corneal opacity and of opaque lines which radiate from the limbus. The patient complains of a feeling of pressure and of a red cloud before the eye. If the injury is limited to the rupture of the walls of Schlemm's canal, the prognosis will be favorable.

Treatment.—Large tears in the sclera, with loss of most of the vitreous humor, and extensive damage to the chorioid and retina, will call for immediate enucleation or the performance of Mules's operation. In case the eye is less severely injured and some vision remains, the surgeon should attempt to save the organ. To this end a general or local anesthetic is used; the eye is carefully washed in a weak bichlorid or normal salt solution; protruding pieces of vitreous, iris, or chorioid are excised; and the lips of the scleral wound are united with sterile catgut sutures. In scleral ruptures concentric with the cornea Nuel's method (Fig. 254) of stitching will be useful. A thread armed with two needles is passed beneath the conjunctiva near the equator and is made to encircle the cornea. When tied, it causes the lips of the scleral wound to approximate. Atropin is then instilled, a bandage is applied, and the patient is confined to bed. If infection occurs, the case will probably require enucleation; if infection does not occur, useful vision may be saved. Intra-ocular tension may rise after the closure of a scleral wound, thus causing secondary glaucoma or staphyloma. The eye may shrink, become tender on pressure, and be a cause of sympathetic ophthalmitis. It is justifiable to attempt for two weeks to save a severely injured eye, since sympathetic inflammation will not occur within that period, and at the end of ten days the intelligent surgeon will be able to foretell the fate of the eye. Exploration of a scleral wound with a probe passed into the vitreous body is dangerous, because of the liability of introducing pathogenic germs.

In some cases of beginning infection the eyeball can be saved by a series of procedures which includes the destruction by fire of the focus of infection. Suppose, for example, a corneoscleral wound has been received, the lens and iris have been injured, the instrument causing the wound has been removed (*i.e.*, there is not a foreign body in the eye), but there is beginning infection of the wound and of the anterior portion of the vitreous body. In such a case the patient should be anesthetized, a large corneal incision should be made, and this should be followed by a large iridectomy, and by delivery of the lens. Then the foci of infection should be destroyed with the electric cautery, iodoform should be placed within the globe, and a compress bandage should be applied. In many instances this heroic treatment will save the eyeball, although vision will generally be lost.

FOREIGN BODIES IN THE SCLERA are not often seen, since such substances are generally propelled with force sufficient to cause them to enter the vitreous chamber. However, pieces of iron, steel, glass, stone, percussion-caps, grains of gunpowder, etc., sometimes lodge in the sclera. If not visible, their location can often be determined by x-ray localization.

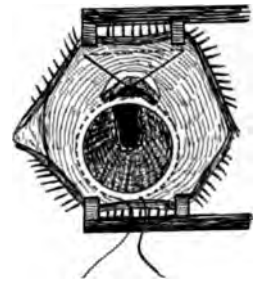


Fig. 254.—Stitch for scleral rupture. (After NUEL.)

In case the suspected foreign body is iron or steel, the sideroscope may be useful in diagnosis, or the Haab magnet can be used. The latter localizes the foreign body by the pain which follows its application.

FOREIGN BODIES IN THE EYEBALL.—If an eye has been injured, the surgeon should try to determine whether the missile has lodged within the globe. The history of the case, the statements of witnesses, and the appearance of the wounding substance (if large) may aid in solving the question. A foreign body may pass through the globe and lodge in the orbit, or it may rest in any of the ocular structures, the vitreous chamber being a favorite location. If the body is loose, its tendency is to sink to the lower and anterior part of the vitreous chamber, adjacent to the ciliary body. While foreign substances may remain within an eye for long periods without exciting inflammation, such a state of affairs is highly dangerous. Owing to its location, to the hemorrhage resulting from the injury, to the opacity of the lens, or to the presence of inflammatory products around the missile, it rarely happens that the foreign body is visible to ophthalmoscopic examination. Consequently its presence can be determined only by an x-ray examination; or, if the substance be iron or steel, by the use of a

giant magnet. It is only in the case of minute splinters, one millimetre or less in diameter, that the x-ray examination may fail to detect a foreign body. Naturally the method employed for the removal of an extraneous substance will be determined by its nature.

Magnetic Foreign Bodies.—If the injury has been inflicted by a piece of iron or steel, which is supposed to have lodged in the eye, the patient should be subjected to the influence of a Haab giant electromagnet at the earliest possible moment. The instrument should be provided with a rheostat. The patient being seated or lying on a table, the eye is anesthetized with cocain or holocain and the tip of the magnet is applied to the eye before the current is turned on. If the metal has entered the eye behind the lens, the tip of the magnet is to be applied to the enlarged wound of entrance. If the original wound is too small to be seen, a scleral opening is to be made with a von Graefe cataract-knife, the incision being made between two of the recti muscles. If the lens is clear, the magnet is to be applied to the ciliary region and later to the cornea. By this method it is possible to draw a foreign body around the periphery of the lens. The presence of the foreign body behind the iris will be indicated by pain and by bulging of the iris. The piece of metal is then to be drawn into the anterior chamber, from which it can be readily extracted through a corneal opening. If the lens is opaque, the magnet is to be applied to the centre of the cornea.

If the use of the giant magnet fails to detect the metal, an x-ray picture should be made. The location and size of the foreign body having been determined, if the substance is magnetizable, the surgeon can choose between two methods, viz.: (1) he can make a scleral incision over or near to the site of the foreign body and then introduce the tip of a Hirschberg, Lippincott, Johnson, or Sweet hand-magnet; or (2) he can bring the front of the patient's eye close to a giant magnet, using this instrument to draw the foreign body forward through the vitreous body, past the zonula of Zinn, and thence through the posterior into the anterior chamber, from which it is to be removed through a corneal section. The first of these procedures will be done under general anesthesia; the second should be accomplished under the influence of cocain or holocain, since the patient's co-operation is necessary to its success. It is not yet time to decide which is the better method. Since few cases occur in which a foreign body, located in the vitreous chamber, is visible ophthalmoscopically, the giant magnet will be the better, in the absence of a sciagraphic examination. The sensation of pain, which follows when the tip of the giant magnet approaches the location of the foreign body, is of diagnostic value. The hand-magnet possesses important advantages over the giant instrument in that it is portable and comparatively inexpensive.

Sweet states that the Röntgen rays offer the most certain method of locating intra-ocular foreign bodies, and he favors the use of this method of examination before resorting to the use of a magnet. On the other hand, Fisher believes that in every case of suspected foreign body (iron or steel)

within the eye, the patient should be immediately subjected to the magnet test. The giant magnet will often aid the diagnosis in two ways: (1) by pain, which is caused by the movement of the foreign body; and (2) by the bulging of the iris, which occurs when a foreign body has been drawn forward through the vitreous humor and around the lens and impinges on the posterior surface of the iris. The early use of the magnet may lessen the danger of infection. The earlier the magnet is used, the better will be the prognosis. If a speculum and fixation forceps are required during the operation, they should be non-magnetic: *i.e.*, made of brass or aluminum. In applying the giant magnet the current is to be turned on gradually until the full force is obtained. If the foreign body does not appear, the current is to be turned off and reapplied with the tip of the magnet in a new position. If a foreign body of large size lies horizontally in the vitreous humor, it will approach the magnet lengthwise, thus obviating the danger of tearing the internal ocular structures.

Fig. 255.—Giant magnet.

Results of Magnet Operations.—While it is impossible to furnish data regarding the use of the hand-magnet, since many unsuccessful cases have not been reported, the statistics of giant-magnet operations are fairly complete. Haab, of Zurich, and Fisher, of Chicago, have recently published their results.

HAAB'S STATISTICS.

Total number of eyes	165
Number of failures	23
Number of eyes from which splinters were extracted	141
Number of eyes requiring enucleation	39
Number of sightless eyes preserved	19
Number of eyes requiring a cataract operation (of these, 51 recovered useful vision)	71

FISHER'S STATISTICS.

Total number of cases	150
Symptoms of metal in eye, but negative result with magnet	49
Metal removed	97
Metal found in eyeball after enucleation—"magnet negative"	4

RESULTS.

Good vision	96
Sightless eyes—"external appearance good"	34
Enucleations	20

Causes of Failure with the Giant Magnet are mentioned by Haab in these words: "Of the whole number of 165 cases in my experience, the operation failed only 23 times. The operation was successful in 141 cases, or in 86 per cent. If we consider the 134 difficult cases in which the splinter penetrated behind the iris and the lens, we find that in these 134 cases the large magnet failed only 23 times to extract the splinter from the eye, and was successful in 111 cases, or 83 per cent. These failures were due to the following circumstances:—

"1. The foreign body was seated too firmly in the back wall of the globe or had pierced it completely.

"2. The splinter was seated in the ciliary body at first or was drawn there by mistake.

"3. The splinter had produced fibrino-purulent exudation, which, according to my experience, greatly hinders its movability.

"4. The splinter had been healed over in the course of months or years."

Removal of Non-magnetic Substances.—These foreign bodies can be located in almost all cases by means of the x-rays. They should be removed with forceps introduced through an incision in the most available part of the sclera.

Operations on the sclera (anterior and posterior sclerotomy) will be described in the chapter on glaucoma.

CHAPTER X.

DISEASES OF THE IRIS.

THE iris is subject to congenital malformations, tumors, inflammations, functional disturbances, and injuries.

CONGENITAL ANOMALIES.

They are coloboma, persistent pupillary membrane, corectopia, polycoria, anirida, ectropion of the uvea, heterochromia, albinism, tumor, atrophy, and adhesion to the cornea.

Coloboma of the Iris may exist alone, but generally is found with a like condition of the chorioid or ciliary body. In most cases the coloboma

Fig. 256.—Congenital coloboma of the iris. (SEGGEL.)

is below, but it has been seen outward, upward and outward, and upward and inward. When the cleft is spanned by a membrane, the condition is called "bridge coloboma." The extent and shape of the defect are subject to great variations. Alone, it does not impair vision, but, being often associated with coloboma of the chorioid and retina, or with microphthalmos, or with coloboma or displacement of the lens, the patient's vision is likely to be much reduced. The cause of the defect is not known. Lang and Collins say that it is not due, as some have supposed, to an unclosed fetal fissure of the iris, since this structure is not developed in two sectors, and the normal fetal iris never has a cleft. The condition is not amenable to treatment.

Persistent Pupillary Membrane is the most frequent of the anomalies of the front of the globe. This membrane, formed from the anterior fibrovascular sheath of the lens, in man should disappear before birth. If a portion remains, the condition named is the result. This anomaly is present in about 1 per cent. of cases. The persistent membrane varies in color, extent, and relationships. The commonest form is a single fine thread, passing from the front of the lens to an attachment on the anterior face

of the iris near its inner circle. There may be several attachments simulating iritic adhesions. The fact that they arise from the front of the iris, which can be determined by oblique illumination, serves to distinguish them from posterior synechiæ. The normal action of the iris is not interfered

Fig. 257.—Persistent pupillary fibres, forming loops. (De Beck.)

with by these congenital tags. The condition is much more often unilateral than bilateral.

Corectopia is an abnormal position of the pupil. Often the pupil is found slightly excentric, but marked displacement is very rare and is usu-

Fig. 258.—Forms of persistent pupillary membrane. (De Beck.)

ally an accompaniment of ectopia lentis. The displaced pupil is often small, irregularly circular or slit-shaped, and inactive. It may be associated with the presence of granular opacity and blood-vessels on the lens, as in the case reported by de Beck (Fig. 259).

Fig. 259.—Forms of corectopia. (De Beck.)

The figure at the left shows granular opacity and blood-vessels on the lens.

Polycoria does not exist in the sense of two or more pupils, each provided with a sphincter, but the term is applied to supernumerary openings in the iris. They are to be explained as gaps occurring from some cause during the development of the eye. Not infrequently, in coloboma of the iris, a band of tissues will be found stretching across the opening, dividing the pupil into two parts.

Aniridia.—Cases supposed to present complete absence of the iris are occasionally seen. The pupil is all black by daylight and all red to the ophthalmoscope. Clinically there is absence of the iris, but dissection and microscopic study of such eyes generally show the presence of the remains of a rudimentary iris. The condition is generally bilateral, and is associated with other congenital anomalies. When aniridia is complete, the entire lens can be seen; when incomplete, the iris is absent at certain points and only segments of the lens are visible. Aniridic eyes are particularly prone to the development of cataract. Vision is often much reduced by dazzling. The wearing of dark glasses is advisable.

Heterochromia is present when one iris presents from infancy a color different from that of the other. It may be symmetrical, or one part of the iris may be markedly darker than the remainder. Small pigment-spots in the iris may be mistaken for foreign bodies by an inexperienced examiner.

Exfoliation of the Iris is a rare condition. Although not congenital, it will be considered here. The process of exfoliation involves the anterior layer of the iris. In a case described by Jackson each iris was divisible into two zones: an upper, where the color was blue-gray, such as is seen in early

Fig. 260.—Polycoria. (De Beck.)

infancy; and a lower, which presented a gray-brown color, the stroma of the iris being seen indistinctly. Between the zones was an irregular strip in which the anterior layer of the iris was hanging in shreds. It is supposed that the change in color in this affection is always from brown to blue or gray. Such eyes are said to be particularly liable to cataract.

Melanoma is a congenital proliferation of the pigment-cells of the iris-stroma, forming a small, dark growth which is of interest by reason of the fact that it may be the starting-point of a melanotic sarcoma. The condition is of rare occurrence.

Ectropion of the Uvea is the name applied to that congenital condition in which chocolate-brown masses, composed of from one to ten nodules, project from the margin of the pupil on to the margin of the iris. They are composed of uveal pigment, which may become detached and float in the aqueous humor. The affection has been incorrectly called papilloma of the iris. It is common in the horse.

Albinism.—A congenital absence of pigment often obtains in the entire uveal tract, as well as in other parts of the body. The condition has been seen in black as well as in white races. The cause of albinism

is unknown. The reason of the failure of the epiblast to form pigment-cells is unexplained. Heredity seems to play little, if any, part in the etiology of this anomaly. The mental and physical condition of albinos compares favorably with that of other persons. According to Gould, the pathologic significance of albinism lies in the fact that the iris is transparent, lack of pigmentation in the chorioid having little pathologic significance except as permitting trans-scleral illumination. Lack of pigment causes the irides to look red; there is photophobia, dazzling, and diminution of vision. Often there is nystagmus. Astigmatism is frequently present and is due in large measure to the pressure of the eyelids (in a state of blepharospasm) upon the globe, the facial muscles also co-operating. Amblyopia is often present in these cases, and strabismus or muscular insufficiency also occurs. Ophthalmoscopic examination usually shows the fundus entirely normal, except for the absence of pigment. The treatment of albinism includes the correction of ametropia and the use of dark glasses.

Congenital Tumors of the Iris are cysts and nævi pigmentosi. Little is known of them. The cysts are mentioned elsewhere in this chapter.

Congenital Atrophy of the Iris is a rare condition which is due to intra-uterine inflammation.

Fig. 261.—Cyst of the iris. (COLLINS.)

The cyst is situated between the two layers of pigment at the back of the iris.

Adhesion of the Iris to the Cornea is a rare anomaly which is usually present in the form of a union between the peripheric portions of these tissues, the central portion of the anterior chamber being, as a rule, of normal depth. Failure of the iris to separate completely from the cornea interferes with the drainage of the aqueous humor and is a cause of infantile glaucoma.

TUMORS OF THE IRIS.

Cysts of the Iris.—These are divisible into serous, or true cysts: epithelial, or implantation cysts, due to the implantation of foreign tissues; dermoid cysts, and entozoal cysts. The true (endothelial) cysts may be congenital or acquired. When congenital they must be accounted for by assuming that a crypt of the iris becomes closed and fluid collects in it. Acquired cysts are nearly all due to trauma, although some are undoubtedly caused by glaucomatous processes. This is particularly true of the cysts situated between the two layers of pigment epithelium on the back of the iris (Fig. 261). Of the implantation cysts much has been written. They may follow a cataract extraction, or any corneal wound which drags epithe-

lium or hairs into the anterior chamber. These tumors present a mother-of-pearl appearance due to cholesterin crystals. They have been called pearl-cysts or cholesteatomata. They contain a lining of laminated epithelium and semisolid contents, consisting of degenerated epithelial cells and fat-globules. Epithelial cells or hair-bulbs transported to the anterior chamber find favorable soil for growth. They may remain quiet for a long time and then become active. When they grow, the eye becomes painful, the tension rises, and a large part of the anterior chamber is filled by the cyst. Unless removed early, the growth may cause iritis, iridocyclitis, secondary glaucoma, or sympathetic ophthalmitis. As might be expected, these cysts can be produced experimentally in the lower animals.

A few cases of congenital dermoid cyst have been reported. Monzon mentions two and Snell, of Rochester, has observed one. The entozoal cysts are chiefly caused by the cysticercus. A few have been caused by the filaria.

DIAGNOSIS.—The diagnosis of cyst of the iris can be made by inspection. The history with reference to trauma should be carefully elicited.

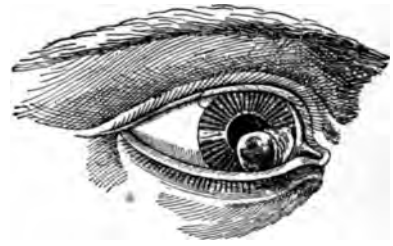


Fig. 262.—Serous cyst of the iris following discission for milky cataract. (AYRES.)

TREATMENT.—The treatment is early and radical removal. A large incision should be made in the cornea with a von Graefe knife, and the part of the iris containing the cyst should be drawn out and excised. The removal must be thorough, otherwise the cyst will recur. Under favorable circumstances vision may be saved. If the eye is already blind, the cyst large, and particularly if the eye is tender to the touch, an enucleation or one of its substitutes will be necessary.

Sarcoma of the Iris.—Of the different portions of the uveal tract, the iris is most rarely the seat of primary sarcoma. Most of the reported cases were melanosarcomata arising from nævi pigmentosi in persons past the thirtieth year, although the disease has been observed as early as the second year by Alt and as late as the seventy-fifth year by Zellweger. In about 25 per cent. of the cases the growth is a lymphosarcoma. The tumor consists usually of spindle cells, some pigmented, others not, arising chiefly from the anterior layers of the iris. It increases by growth of the stroma or adventitia cells, fills the anterior chamber, and involves the ciliary body. The most noticeable of the early symptoms is the presence of a growing tumor, which is usually pigmented. It may exist for months or even years

before causing inflammatory symptoms, or before interfering with the movements of the iris. The tumor is often vascular and nodular. It may cause hemorrhages into the anterior chamber. The lens becomes displaced, the tension increased, iritis develops, and the eye becomes blind. These growths are said by Kerschbaumer not to pierce the corneoscleral region often, thus differing from tubercle of the iris.

DIAGNOSIS.—Sarcoma of the iris may be mistaken for simple melanoma, gumma, or tubercle. The symptoms have been carefully studied by Veasey, who collected forty-six undoubted cases of sarcoma. From Veasey's article and other sources the author has constructed the following table:—

MELANOMA.	GUMMA.	TUBERCLE.	MELANOSARCOMA.
Becomes darker and darker.	When a gumma appears there is severe iritis.		Retains its primary shades usually.
Is congenital.		Appears usually in persons under twenty.	Is not congenital.
No specific history.	History and symptoms of syphilis.	Tubercular foci often found elsewhere.	
		Inflammatory symptoms appear early.	No inflammatory symptoms in early stage.
	Color: iron-red or deep yellowish red.	Color: yellowish white, light grayish white, or light grayish yellow.	Color: reddish gray, blackish, light brown, or flesh-color.
	Non-vascular.	Non-vascular, as a rule.	Is vascular.
		Irregular in form.	Rounded in form.
Antisyphilitic treatment does not influence it.	Antisyphilitic treatment removes it.		Antisyphilitic treatment does not influence it.
Is stationary.		Is of rapid growth.	Grows slowly.
	Is generally situated peripherally.	Is generally situated near the pupil.	May have multiple points of growth.
	Hypopyon may be present.	Hypopyon may be present.	

TREATMENT.—In view of the difficulties attending the diagnosis of iridal growths, it will be advisable to operate upon them early, making a broad iridectomy which shall include all of the mass and a part of the healthy iris on each side. Then a microscopic examination can be made and the nature of the growth determined. If it proves to be a sarcoma, the question of enucleating the eye must be considered, although it will be advisable to wait for a time and to note the effect of the iridectomy. In a few cases not only has the eyeball been saved and useful vision preserved, but no recurrence has been noted for years after the operation. The growth of an iris sarcoma is slow, and a period of at least three years must elapse before a case treated by iridectomy can be considered as cured. If vision is seriously affected, an immediate enucleation should be performed. If, after iridectomy, the growth shows signs of recurrence, the eye must be removed immediately. Cases of sarcoma of the iris cured by iridectomy have been reported by Arlt, Kipp, Knapp, Veasey, and others. On the other hand, it may be impossible to determine whether the ciliary body is involved. Wood and Pusey, who have given the latest contribution on this subject, hold that an enucleation should be made as soon as the diagnosis of iris sarcoma has been established.

Vascular Tumors of the Iris.—These have been seen a few times as dark, lobulated growths, springing from the iris and bleeding spontaneously. In some cases they are associated with multiple nevi scattered over the whole body. Vascular iridal tumors are likely to be mistaken for sarcomata. They should be removed by making a broad iridectomy.

Unusual Tumors of the Iris.—In addition to the growths already described, the iris is in rare cases the seat of myomata, myosarcomata, lymphomata, and of certain epithelial formations resembling carcinomata. The nature of these growths can be determined after removal. *Lymphomata* are found in cases of splenic leukemia. The growths may be situated deeply or superficially in the iris-tissue. When superficial, they are present as multiple gray, transparent nodules surrounded by a vascular network. When deeply placed they cannot be seen, but their presence should be suspected because of chronic iritis with pupillary exudate and vitreous opacities. Unlike tubercles, lymphomata of the iris do not suppurate. They may be confounded with gummata. The disease is found chiefly in young persons, and, according to Horner and von Michel, it precedes the general ma ni-

Fig. 263.—Cysticercus which has broken through an iridal vessel. (KRAEMER.)

festations of leukemia. Microscopic examination shows a mass of le co-cytes in the stroma of the iris, which is more dense than normal and pres nts vessels gorged with white corpuscles. In benign forms the neoplasm ay entirely disappear, leaving the iris partially discolored and atrophic, w ile grave cases end in total atrophy of the globe. Aside from the use of atro in, local treatment is not indicated in these cases.

Leprosy of the Iris.—According to Neve, the iris is often attacke in ocular leprosy, especially where the infiltration has begun in the cor eal margin. Nodules form in the iris-angle, partially obliterating the ante ior chamber. Less often a single nodule is present, but seldom is on or r ear the edge of the iris. The whole iris-tissue becomes softened by a chr nic inflammatory process. There is a deposit of fibrin which occludes the pu pil.

Tubercle of the Iris.—When tuberculosis of the iris (Fig. 3, Plate X II) assumes the solitary form, iritis may be absent for a considerable time. S uch cases were described by von Graefe under the name granuloma of the ris. The tubercular nature of such growths was first demonstrated by Haab. he differentiation between this disease, gummatous iritis, and sarcoma may be difficult. Tubercle is generally found between the fourth and twenty-f rst

years, while sarcoma usually appears later in life. The history of the case and the finding of tubercular foci elsewhere will aid in diagnosis. Tubercle is of more rapid growth than sarcoma (Andrews). In any case of solitary mass in the iris antisyphilitic treatment should be instituted. If it fails, the mass should be excised by iridectomy and examined microscopically and by inoculation experiments on rabbits or guinea-pigs. If the entire mass cannot be removed, an enucleation should be made, and in all cases of multiple growths the eye should be excised (Bull). Internal treatment with creosote, and injections of tuberculin, may be tried in the early stages of iridal tuberculosis before resorting to enucleation. Koster has recently reported cures from the injection of air into the anterior chamber. The chamber is first partly emptied; air is then injected through a Pravaz syringe. Patients with tubercular iritis often die from tubercular meningitis.

Cysticercus.—The parasite appears in the anterior chamber as a round, whitish, grayish, or yellowish mass, about one millimetre in diameter. At one particular part of the cyst a whitish projection is noticed which changes its shape from time to time. The mass may be found in the bottom of the anterior chamber or it may float free in the aqueous. At the time it breaks forth from an iris vessel there is great pain. The parasite should be removed through an incision made in the cornea.

Filaria. — The filaria has occasionally been found in the anterior chamber or iris of persons living in the Orient or in Africa. The disease is not seen in England and America except by importation.

INFLAMMATION OF THE IRIS.

Iritis is an inflammation of the iris. It may be congenital or acquired, idiopathic or traumatic, primary or secondary, acute or chronic, simple or complicated. Since the iris and ciliary body receive the same blood-supply, both are usually involved in inflammation simultaneously. For many reasons, however, it is advisable to consider their diseases separately. Iritis is a subject of the greatest importance to the patient, to the general practitioner, and to the ophthalmologist. Recognition of the disease and proper treatment will give satisfactory results. Unfortunately, however, mistakes in diagnosis often occur, and in consequence the patient becomes partly or entirely blind.

Symptoms.—In idiopathic iritis the subjective signs are pain, photophobia, and loss of vision. The objective signs are lacrimation, congestion of the corneoscleral zone, immobility or sluggishness of the contracted iris; change in its color, thickness, and form; and, under a mydriatic, the presence of adhesions. These either bind the iris to the capsule of the lens posteriorly, making the pupil irregular in form, or to the cornea in front. Under mydriasis the pupil may be round, in which case careful examination will show deposits of pigment on the lens-capsule. Pain in iritis is referred to the eye, the nerve-exits around the orbit, the temple, or, as will often be

found, to the side of the nose along the course of the nasal nerve. Pain in iritis is often worse at night or in the early morning. It may be entirely absent, or may be so excruciating that the patient is clamorous for relief. The eye is tender on pressure, particularly at a point mentioned by Callan, about two millimetres behind the corneoscleral junction, under the middle of the upper lid. Loss of vision is great in those forms of iritis in which the aqueous humor becomes turbid or where exudation into the pupillary area is considerable. Accommodative power is also impaired.

On inspection, the iris is seen to have a muddy look, its lustre is absent, and its tissue appears swollen. Often the distended minor arterial circle will surround the pupil like a ring. The change in color, due to hyperemia and

Fig. 264.—Pericorneal injection in iritis. (Author.)

(Original drawing by Dr. R. W. Mills.)

inflammatory products, causes a blue iris to appear greenish and a brown one to look yellowish. The pupil is small, and reacts to light slowly or not at all. The zone of redness around the cornea is deeply seated, as in any serious inflammation of the anterior segment of the globe. There is photophobia and abundant lacrimation. If these symptoms are present, and the tension of the eye is not increased, the practitioner will be justified in the use of a mydriatic. A 1-per-cent. strength solution of atropin or a solution of scopolamin of the strength of $^1/_5$ of 1 per cent. is to be used. Half an hour after dropping the solution into the eye the pupil will appear dilated irregularly, if adhesions between the iris and lens-capsule are too strong to be broken by the mydriatic; or, as often occurs early in the disease, the pupil

will be round, and light concentrated on the lens by a glass will show minute pigment-spots marking the site of the ruptured adhesions. After a slight iritis these spots will be found, since the posterior layer of the iris is the pigment layer. Inspection of the anterior chamber may show a turbid aqueous; the cornea may be steamy.

Diagnosis.—Iritis is often mistaken for acute catarrhal conjunctivitis. The diagnosis can be made by attention to the following points: Conjunctivitis presents a discharge; uncomplicated iritis does not, but generally in iritis there is an accompanying conjunctivitis. In conjunctivitis the greatest redness is situated posteriorly, where the ocular and palpebral parts of the conjunctiva join; in iritis the greatest redness is around the corneo-scleral junction. The redness of iritis is more deeply placed than is that of conjunctivitis. The injected conjunctival vessels are distinctly seen, while the deeper vessels of the circumcorneal zone present a deep, diffuse-red color.

The iris in conjunctivitis responds to light. In early iritis it responds sluggishly, if at all; more often it is immobile and contracted. Vision is

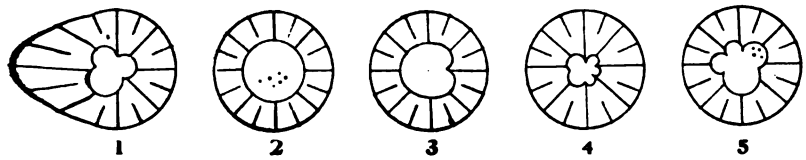


Fig. 265.—Different appearances of the pupil in iritis, after the use of atropin.

1, Three adhesions. *2*, Pupil dilated regularly, with pigment-dots on the anterior capsule of the lens. *3*, One adhesion. *4*, Multiple adhesions with little dilation. *5*. Adhesions at four points and pigment-dots showing where the mydriatic has broken up an adhesion.

not affected in conjunctivitis, barring the spreading of mucus over the cornea, the existence of a complicating keratitis, or the presence of a corneal scar; in iritis vision is usually much reduced from turbidity of the aqueous humor or from exudation on the lens. The tension of the eye is not changed in conjunctivitis; in iritis there is occasionally increase of tension in the height of the disease. The color and lustre of the iris are not changed in conjunctivitis; in iritis the iris looks muddy, has lost its bright appearance, and is often changed in color. In conjunctivitis the pupil dilates regularly after the use of a mydriatic; in iritis the pupil may dilate regularly or irregularly, due to causes explained above.

Differentiation between Iritis and Glaucoma.—It might be supposed that diseases as unlike as iritis and glaucoma should not be confounded, but it often occurs that the practitioner considers a case of acute inflammatory glaucoma to be iritis, and treats the glaucoma with atropin—the worst possible treatment. Both diseases reduce vision and present pain as a prominent symptom. The following table gives the points in differential diagnosis:—

IRITIS.	GLAUCOMA.
The pupil is small.	The pupil is dilated.
Usually the patient is under forty-five years of age.	Usually the patient is over middle age.
Tension may increase in the height of the disease.	Tension is increased, either intermittently or permanently.
The anterior chamber is of normal depth except in neglected cases with complete annular synechia; in these it is deeper than normal.	The anterior chamber is shallow.
The cornea is sensitive to the touch.	The cornea is anesthetic.
The ophthalmoscope cannot be used with satisfaction.	The ophthalmoscope often will show excavation of the head of the optic nerve and pulsation in the retinal arteries.
The chief causes of iritis are trauma, syphilis, rheumatism, and gonorrhea.	The causes of glaucoma are numerous, including closure of the iris-angle, trauma, intra-ocular growths, and certain conditions of the blood and blood-vessels.
The refraction has no etiologic bearing.	Refraction generally is hypermetropic.

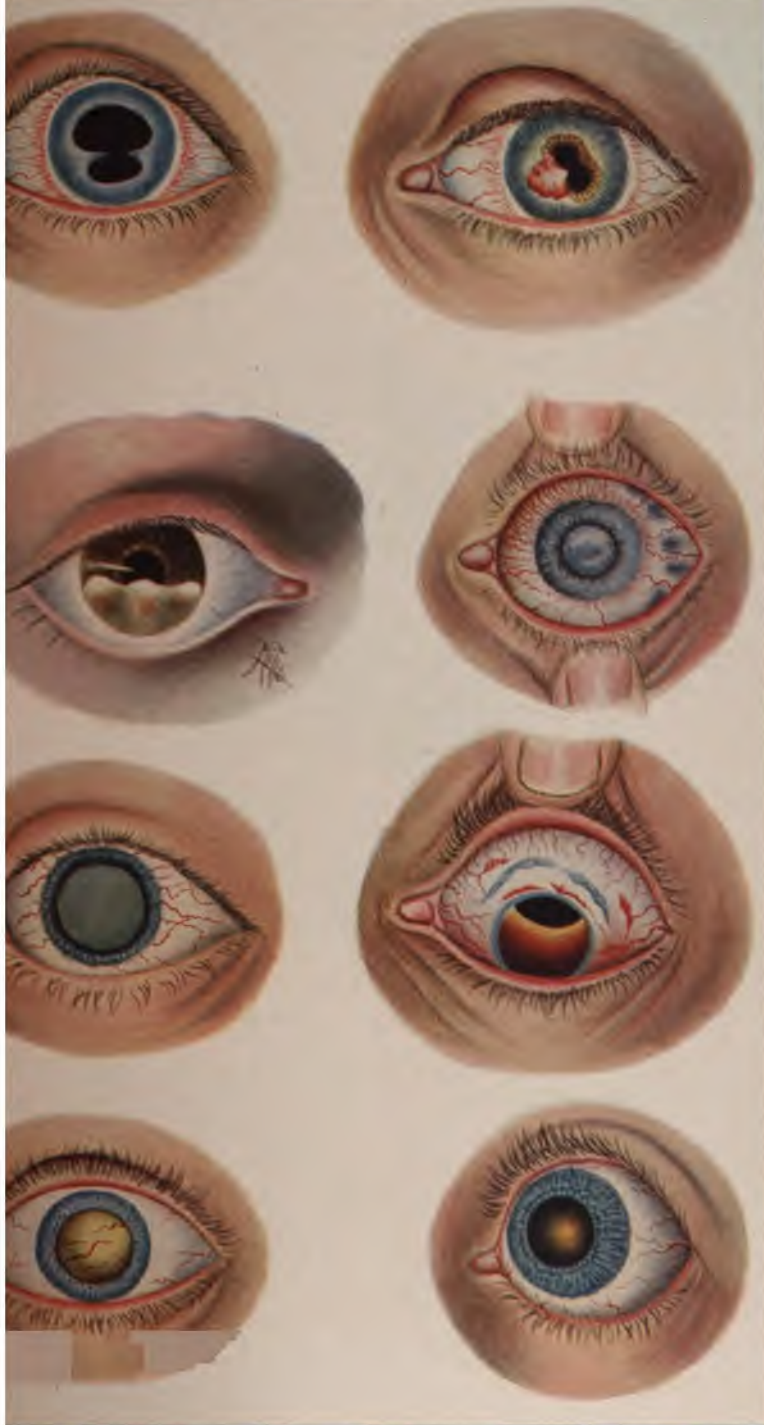
Differentiation between Iritis and Cyclitis.—While pathologic study shows involvement of the ciliary body in nearly all cases clinically diagnosticated as iritis, it is not customary to speak of the condition as a cyclitis or an iridocyclitis unless certain symptoms are present. These include: (1) edema of the upper eyelid, which is not present in simple iritis; (2) alteration of intra-ocular tension, which may be increased or diminished; (3) the presence of considerable pain or tenderness when pressure is made in the region of the ciliary body; (4) the presence of deposits on Descemet's membrane; (5) greater loss of vision than can be accounted for by the muddiness of the aqueous humor.

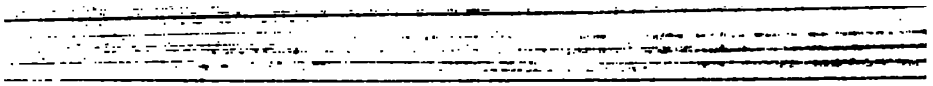
VARIETIES OF IRITIS.—Having made a diagnosis of iritis, it will be in order to determine which variety of the disease is present: serous, plastic, or parenchymatous.

Serous Iritis (Fig. 1, Plate XII), so called, is characterized by a deep anterior chamber, the presence of deposits in the aqueous and on the back of the cornea, and a mildness of the subjective symptoms. This disease may be caused by syphilis. It is the form of iritis often found in sympathetic ophthalmitis. Often the pupil is dilated and the tension of the eye is increased by physical and chemic changes in the aqueous, thus simulating glaucoma. Inasmuch as this disease has been shown by recent pathologic studies to depend on alterations in the glands of the ciliary body, it will be further discussed under the name "Serous Cyclitis."

Plastic Iritis is the type of iridal inflammation which is most frequently encountered. It is called *plastic* from its tendency to form adhesions between the iris and crystalline lens, and to occlude the pupil. It may run an acute, subacute, or chronic course. It presents the classic symptoms of iridal inflammation: pain; pericorneal injection; immobility of the iris,

PLATE 12





which is discolored; and posterior synechiæ. Aside from the ordinary attachments existing between the iris and lens, plastic iritis may result in the pouring of an exudation into the anterior chamber, filling the pupillary area with a gelatin-like mass *(fibrinous,* or *spongy, iritis).* Syphilis is a frequent cause of plastic iritis, the involvement of the iris occurring generally between the second and ninth months after the appearance of chancre. Exceptionally iritis in a syphilitic subject may be delayed till the eighteenth month. Both eyes are generally involved, but usually not at the same time. Rheumatism is a frequent cause of plastic iritis.

Fig. 266.—Gummatous iritis. (Author.)
(Original drawing by Dr. R. W. Mills.)

Parenchymatous Iritis is divisible into suppurative, gummatous, and tubercular forms. In all forms of parenchymatous inflammation the iris is much thickened and distorted.

In *suppurative iritis* pus-cells are present in the tissue of the iris, and hypopyon is common. The most frequent cause of suppurative iritis is infection by a wound. The hypopyon of the idiopathic form of suppurative iritis disappears rapidly, since it consists only of pus-cells, and is not mixed with fibrin as is the hypopyon of ulcus serpens corneæ (Fick). Diabetes and acute infectious diseases, such as pneumonia, cerebro-spinal fever, influenza, etc., are causes of suppurative iritis. It also occurs from embolism in puerperal septicemia and pyemia.

Gummatous Inflammation of the Iris (Fig. 2, Plate XII) appears in the later secondary stage of syphilis, and presents clinical signs peculiar to itself. In rare instances it is found in infants with hereditary syphilis. The iris shows one or more yellowish-brown or reddish-brown nodules, varying in size from a pin's head to a pea. They are found in the ciliary or pupillary border or midway between the two, and are often crossed by vessels. Although found in the secondary stage of syphilis, the name gummata has been applied to them. They soon disappear under treatment without leaving scars in the iris-tissue. Some authors apply the term *iritis papulosa* to this condition, and reserve the name gummatous iritis to those cases of true gummata which appear later in the history of syphilis.

Tubercular Iritis is due to the development of the tubercle bacillus in the iris. It is secondary to a tubercular focus located in another part of the body, and is found principally in persons under twenty years of age. Direct tubercular infection of the iris can occur only through a wound. In

Fig. 267.—Iritis with hypopyon and chemosis. (Von Ammon.)

The pupil is irregular. The collection of pus reaches to the lower part of the pupil. The chemotic conjunctiva forms a ring around the cornea. The vessels of the conjunctiva are much enlarged.

this form of iritis small, gray, transparent nodules are seen which slowly change, some disappearing while others form. The disease is often bilateral. Recovery may take place, but the eye is generally lost by tubercular iridocyclitis. The solitary tubercle of the iris has been described with the tumors of this part.

Pathology.—Iritis causes various changes in the eye according to *(a)* the nature of the inflammation and *(b)* the duration and treatment. In the simple plastic form an exudation of fibrin takes place into the tissue of the iris itself, and into the anterior and posterior chambers. A few pus-cells are found in the exudate. The aqueous humor is made muddy by the exudate and the pupil appears grayish in consequence. The particles of exudate are deposited in the bottom of the anterior chamber and form an hypopyon. If the hyperemia is great, some of the vessels rupture and the blood also sinks to the bottom. A layer of exudate is also found on the anterior surface of the iris, giving it a blurred appearance. This layer may glue the iris to the back of the cornea, as shown in Fig. 268, or, as more

often happens, it simply fills up the pupil (occlusion of the pupil), obstructs vision, and leads to the formation of opacity in the lens. Exudation into the posterior chamber causes adhesions (posterior synechiæ) between the iris and the anterior capsule of the lens. The adhesions develop chiefly at the pupillary margin, the point where normally the iris and capsule are separated by a very thin water-bed. It may happen that the exudate occupies the whole space between the iris and the lens, thus making a complete posterior adhesion. In the formation of adhesions, it is not the iris-stroma, but the pigment layer, which becomes attached to the lens. In response to a mydriatic the iris is drawn toward the periphery, exposing the pigment. If the adhesions are completely severed, a round pupil results, and pigment-spots remain on the anterior capsule. In case the whole pupillary margin

Fig. 268.—Antero-posterior section of an eye which shows post-iritic glaucoma, calcareous cataract, and chorioiditis. (AUTHOR.)

These changes were all caused by a neglected iritis. In the fresh specimen the iris, through a large part of its extent, was adherent to the posterior surface of the cornea, from which it has been separated in order the better to show the annular posterior synechia.

of the iris becomes adherent (annular posterior synechia), the posterior is completely shut off from the anterior chamber. Thus, the passage of aqueous humor into the front of the eye is stopped and the iris becomes bulged forward *(iris bombé)*, producing the condition known as post-iritic glaucoma. If the process is not checked by an excision of part of the iris, all of the tissues of the eyeball become involved, the optic-nerve head undergoes excavation, and vision is destroyed.

During an attack of iritis many other tissues suffer. The cornea is involved in every case (Friedenwald), being more or less opaque owing to infiltrations in the substantia propria or to deposits on Descemet's membrane. In many instances the corneal opacity can be detected only by careful examination. Iridocyclitis, iridochorioiditis, hyalitis, and neuroretinitis are frequent complications. As a result of frequent attacks of

acute iritis, or from a long-continued chronic state, the iris becomes atrophic. It has a bleached appearance, the normal delicate markings are lost, and the pupillary margin looks frayed.

ETIOLOGY.—Iritis may be the result of trauma, but generally it is due to a constitutional vice. Syphilis causes about 50 per cent. of the cases. Rheumatism is another frequent factor, producing about 30 per cent. Other causes are anemia, malaria, diabetes, gonorrhea, gout, neoplasms, and acute febrile diseases. Diabetic iritis is one of the rarest forms. Sympathetic ophthalmitis causes the form of iritic and cyclitic inflammation which will be described as "Serous Cyclitis." In many cases of iritis it is impossible to determine the cause, since there is no constant symptom by which to recognize the diathesis present. If syphilis always produced the characteristic nodules in the iris, recognition would be easy; but many cases undoubtedly due to syphilis do not present symptoms and exudation different from those which are found in rheumatic iritis. Rheumatic iritis has long been credited with a marked tendency to recurrences, but it is not certain that this statement will bear investigation.

Iritis occurs chiefly in young adults and in middle-aged persons. In children it rarely occurs, and is then due usually to hereditary syphilis, or tuberculosis, or exists as a complication of corneal ulcer. In proof of the rarity of iritis in syphilitic infants it may be stated that George Carpenter, of London, although he has examined the eyes of hundreds of such subjects, never saw an undoubted case: *i.e.*, one with irregular pupil under atropin; and Jonathan Hutchinson, Sr., records only twenty-three cases. Of Hutchinson's cases, the youngest was six weeks, the oldest sixteen months of age. Iritis is sometimes found in young girls at the period of puberty.

Traumatic iritis may arise from blows, operative procedures, penetrating wounds, or the lodgment of foreign bodies. It may be plastic or purulent; if the latter, it is a part of panophthalmitis. Perforating corneal ulcers also cause iritis. An extremely severe and long-continued inflammation of the iris follows the penetration of the hairs of the caterpillar into the conjunctiva, cornea, and iris, producing the condition known as ophthalmia nodosa. This form of ocular inflammation has been described in the chapter on the conjunctiva.

It is because of the impossibility of founding a classification of iritis on the etiology that the author has adhered to the old division into serous, plastic, and parenchymatous forms.

Congenital Iritis, as shown by the presence of congenital posterior synechiæ, has been observed by Bull in infants within two hours after birth. The disease is to be attributed to syphilis, and this opinion is confirmed by the occurrence of "snuffles" within a few weeks after birth, and by the finding of mucous patches around the anus. Owing to the absence of ciliary injection and the infrequent occurrence of discoloration of the iris, iridal inflammation in infants may be overlooked. In fact, it can generally be detected only by the use of atropin and oblique illumination.

Diabetic Iritis is a rare form of ocular disease. Galezowski met with it 7 times among 144 diabetic patients with ocular diseases. Leber saw it 9 times among 39 diabetics. This type of iritis is often purulent, and a noteworthy fact is the rarity of purulent iritis without the coexistence of purulent keratitis. The iridal inflammation is rarely of a severe grade, and hypopyon, when present, is usually small. The pupillary space is likely to be filled with a fibrinous membrane, which disappears rapidly under appropriate treatment. Occasionally the disease assumes the serous type. While the course of diabetic iritis is generally favorable, there may be complications, such as cyclitis, chorioiditis, cataract, and opacities in the vitreous body. Iritis, following the operation of iridectomy, is not infrequent in

Fig. 269.—Proliferation of the pigment layer of the iris in diabetes. (AUTHOR.)

(Photomicrograph by DR. H. P. WELLS.)

diabetics. Microscopic examination has shown the frequent occurrence of changes in the pigment-epithelium layer of the iris, ciliary body, and retina. They include a loosening, proliferation, and probably edematous swelling of the pigment-cell layer on the posterior surface of the iris. Hence, following an operation such as iridectomy or cataract extraction, the aqueous humor may appear black from pigment held in suspension. Cases of diabetic iritis require treatment for the constitutional condition in addition to the usual local remedies.

Senile Iritis.—In elderly persons it is not unusual to find that iritis is overlooked by the medical attendant because of absence of the common signs. There may have been an acute catarrhal conjunctivitis involving both eyes. After the application of the usual treatment one eye recovers

and the other does not. Inspection shows no muddiness of the iris or aqueous humor, the pupil responds to light, tension is normal, pain is slight, and perhaps the only sign of iritis is a slight pinkish redness around the cornea. After excluding glaucoma by ophthalmoscopic examination, the surgeon should dilate the pupil *ad maximum* with atropin. Then the presence of adhesions, either at the pupillary margin or more frequently at the peripheral part of the iris, will declare the true condition. Continued use of a mydriatic will cure these cases. During the treatment the tension should be watched. Hale has called attention to two signs of diagnostic value in these cases of quiet iritis: *(a)* one eye is usually involved more than the other and *(b)* the muco-purulent discharge characteristic of conjunctivitis disappears and is succeeded by profuse lacrimation. The author has seen several of these cases which he attributed to malaria.

Uvéite Irienne.—Grandclément has called attention to the importance of distinguishing from ordinary iritis those cases in which only the posterior layer of the iris is involved. To these he applies the term ***uvéite irienne.*** In every respect this affection is different from ordinary iritis, and the points in differentiation are given in the following table:—

Uvéite Irienne.	Iritis.
Occurs almost exclusively in women.	Occurs irrespective of sex.
Both eyes are always affected.	Often is unilateral.
Disease lasts for years and shows slight periodic exacerbations, lasting five or six days.	Symptoms are violent, and the disease is cured in six or eight weeks.
Causes are unknown.	Causes are trauma and constitutional vices.
Only effective treatment is iridectomy.	Proper treatment is the use of mydriatics.
No discoloration of anterior surface of iris.	Anterior surface of iris is discolored.
Pain is almost absent and redness is slight.	Pain is severe and redness is marked.
Occurs about the time of the menopause.	

Prognosis of Iritis.—The prognosis will depend on *(a)* the time of recognition of the disease, *(b)* the treatment instituted, and *(c)* the character of the cause. An eye whose iris has been the seat of inflammation rarely makes a complete recovery, although vision may be retained and the eye remain quiet through life. Simple plastic iritis, if recognized early, offers a favorable prognosis. The parenchymatous forms, except when due to tubercle, usually give fair results. Suppurative iritis generally ends in panophthalmitis and the loss of the eye. In serous iritis due to sympathetic ophthalmitis the prognosis is very grave. Iritis following ulcers of the cornea is a serious condition, and is discussed in the chapter on "Diseases of the Cornea." If seen early and properly treated, the great majority of cases of idiopathic iritis will give satisfactory results. The occurrence of

a pronounced cyclitis and chorioiditis adds much to the gravity of the prognosis. Post-iritic glaucoma is a formidable condition which often produces symptoms demanding enucleation of the eye.

TREATMENT.—The sovereign remedy for iritis is the strongest mydriatic that the patient can bear. A solution of atropin (1-per-cent. strength) or of scopolamin ($^1/_5$ of 1-per-cent. strength) will break up the adhesions when used early in the case, and relief of pain and hyperemia will follow this treatment. Scopolamin is particularly valuable in the parenchymatous and traumatic forms of iridal inflammation. The mydriatic should be used three or four times a day for the first week or ten days, after which period it will suffice to use it once a day. The manner of making the instillation is of importance. The solution should be gently warmed and dropped on to the upper middle part of the sclera while the eye is directed downward, thus causing the solution to float over the cornea and promoting absorption. In delicate nervous individuals, and particularly in females and children, care must be taken not to produce the toxic effect of the drug. Of the two mydriatics mentioned, scopolamin is the more powerful and the more likely to cause toxic symptoms. It is advisable either to compress the puncta while using the drops or to have the patient incline his head in such a direction that the medicine will flow toward the outer canthus. In case the atropin solution causes conjunctivitis, eczema of the lids, and toxic symptoms, the alkaloid can be used in the form of an ointment (atropin, gr. iv; cocain, gr. vj; vaselin, ℥j), or one of the ophthalmic discs containing atropin and cocain can be placed under the lower eyelid and be permitted to dissolve. The latter method is clean and efficient.

Darier claims that dionin aids the mydriatic action of atropin, hastens absorption of pupillary exudations, diminishes intra-ocular tension, and relieves pain. He uses the following collyrium:—

℞ Dionin ..... gr. iss.
Cocain ..... gr. iss.
Atropin ..... gr. ss-$^5/_4$
Water ..... ℥iiss.

M. Sig.: One drop to be placed in the affected eye six to eight times a day.

Unfortunately it often occurs that patients come after adhesions have bound the iris to the lens-capsule. An attempt must be made to secure dilation of the pupil, and this will necessitate the forced use of mydriatics with the simultaneous internal administration of considerable amounts of mercury. Absorption of the mydriatic will be facilitated by the previous local use of a few drops of adrenalin solution (1 to 5000), or by the employment of cocain simultaneously with the mydriatic. Then, by dropping the scopolamin or atropin solution on to the outer part of the cornea at proper intervals, the puncta being compressed and the constitutional symptoms carefully watched, the maximum effect of the mydriatic may be ob-

tained. Even if full dilation is not secured, there may be sufficient enlargement of the pupil to permit the normal flow of the aqueous humor.

The patient's general condition must be carefully studied, and here the family physician and the ophthalmologist should work together. It is of importance that any constitutional vice should be recognized and treated. Search should be made for syphilis, rheumatism, gout, malaria, diabetes, and tuberculosis. In diabetic and rheumatic iritis great benefit follows the use of large doses of salicylate of sodium. In gummatous iritis huge doses of iodid of potassium are often required. Attention should be directed to the condition of the intestinal tract. It is a common observation that patients with iritis feel much better after the intestinal tract has been evacuated. Hot-air baths are of use in hastening the absorption of the inflammatory products. The condition of the nose and throat should be investigated. If the patient has a "cold," or is suffering with chronic hypertrophic rhinitis, great benefit will follow the proper treatment of these conditions. The patient should be given a pair of dark glasses to protect the eyes, and, as soon as the acute symptoms have subsided, should be ordered to get out of doors every day. Under no circumstances should the inflamed eye be bandaged, except in traumatic iritis. In case no constitutional dyscrasia can be detected, the internal use of laxatives, salines, and mercurials will still be beneficial. Often in anemic subjects the administration of quinin or Warburg's tincture will be advantageous. Stimulants are generally out of order in iritis.

Pain, during the acute period, may call for the internal administration of chloral or the hypodermic injection of morphin after the local use of dry heat has failed. A convenient method of applying dry heat is the use of the Japanese hot box. A valuable remedy for pain is hyoscin (gr. $^1/_{100}$ at bedtime). Recently dionin, in 2-per-cent. strength solution, has been successfully employed to relieve the pain of iritis. The use of cloths wrung out of boiling hot water, applied to the eyelids for a few seconds, is often acceptable. The use of leeches, ointments, blisters, poultices, and setons is valueless and in some cases is positively harmful. Desmarres's depletion —*i.e.*, the section of a subconjunctival vessel, which is permitted to bleed freely—often will relieve pain and check the advance of the inflammation. If pain is severe and the tension is increased, paracentesis of the anterior chamber should be done, removing 2 or 3 drops of aqueous humor. This little operation gives relief, and can be repeated every other day until pain and tension are diminished. Paracentesis is to be done under holocain anesthesia; the puncture is to be made with the von Graefe cataract-knife, the back of the blade being placed toward the iris, or the stop-keratome can be used. The use of cocain as a routine practice in iritis is not to be advised. The mydriatic must be continued until all signs of redness have disappeared. It is important that the patient should not apply himself to any near work until several weeks after he is entirely well, since too early use of the accommodation may cause a recurrence of the inflammation.

In iritis produced by infectious diseases atropin locally and the proper internal treatment must be employed. Often in these rare cases the use of quinin and stimulants will be necessary.

After pain and redness have disappeared the surgeon should note the damage done by the disease. If adhesions have shut off the anterior from the posterior chamber, an immediate iridectomy should be made. It will give a good result provided the ciliary body and retina are intact. In recurrent iritis, with numerous adhesions, an iridectomy should be made between the attacks and will often prevent recurrences, provided the general system is cared for. If the pupil is blocked by exudation interfering with vision, an optical iridectomy is of value.

Before resorting to an excision of the iris for extensive adhesions following iritis, the surgeon should determine, as far as possible, the condition of the deep parts of the eye, the acuity of vision, the effect of mydriatics upon the iris, and the condition of this membrane. If the iris is atrophic or the vision is greatly reduced, the effect of an iridectomy will be slight or *nil*. The excision of an atrophic iris is a difficult matter. If the tension of the eye is subnormal, it is probable that no good will follow the iridectomy. On the other hand, increased tension remaining after recovery from acute iritis renders iridectomy imperative, and here the operation is often followed by brilliant results. When only the pupillary margin is attached, an iridectomy can be made easily; while if the whole iris is bound down to the capsule, the excision is difficult. After completing the operation the surgeon may find that he has removed only the anterior layers of the iris, while the pigment layer remains attached to the capsule. In such cases it is often necessary to extract the lens, together with a large part of the iris, to get results. Even under the most skillful manipulation some of these cases will go to destruction, either by hemorrhage during or following the operation, by infection, or by iridocyclitis.

Where only few posterior synechiæ are present, they do not call for treatment. It has been held that their presence tends to produce relapses; this, however, is doubtful, since every ophthalmic surgeon has met with patients presenting extensive posterior synechiæ, who for many years remained free from a second attack of iritis.

The operation for detaching such adhesions (corelysis), so often practiced by the older ophthalmologists, is now obsolete. The condition of the refraction after iritis demands attention. During the attack the emmetropic eye becomes myopic, the hypermetropic less so, and the myopic eye more myopic. This condition gradually disappears and the refraction returns to its original state. On account of the existence of this transient myopia it will be advisable to test the refraction several times during the year following recovery from iritis.

Treatment of Uvéite.—In the form of inflammation which Grandclément has described under the name *uréite irienne* atropin is of little value. Such cases should be treated by iridectomy.

INJURIES OF THE IRIS.

On account of its protected position the iris rarely suffers from trauma without other tissues being involved. However, it may happen that the injury of the iris is of greater moment than is that of other parts.

Iridodialysis, the most common of these conditions, is a tearing of the iris from the ciliary body. It often follows lacerated wounds and contusions, or may occur during operations on the iris, owing to unskillful manipulation or to rebelliousness of the patient. It is always accompanied by hemorrhage into the anterior chamber (hyphemia). The blood may conceal the lesion for several days. The rupture may be very minute or large; or the whole iris may be torn from its attachments (traumatic irideremia), and be extruded from the eye or drop to the bottom of the anterior chamber, where it remains as a shriveled, grayish mass. If the rupture is small, vision is only slightly or not at all affected; if large, the patient will complain of monocular diplopia and dazzling. This is particularly true if the rupture is not covered by the upper lid. Idiopathic iridodialysis may result

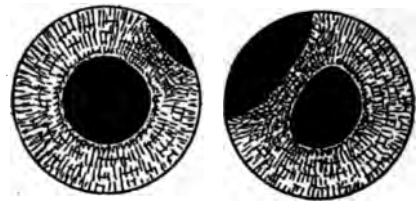


Fig. 270.—Iridodialysis. (De Beck.)

from the growth of a tumor pushing the iris from its attachment. In traumatic iridodialysis, under retinoscopic examination, a red reflex from the fundus shows through the rent. In iris-detachment from tumor it is absent.

Treatment.—The eye should be protected from bright light and atropin should be frequently instilled. It has happened a few times that the iris has become reattached under this treatment. If the laceration is extensive, the operation known as iridenkleisis, which is advised by Eugene Smith, of Detroit, can be performed. In this procedure the cornea is opened with a keratome and the torn iris is dragged into the wound and held *in situ* between the lips of the incision. Smith states that this operation will not cause sympathetic ophthalmitis.

Anteversion of the Iris may follow iridodialysis, the iris being twisted upon itself so that the pigment layer shows in front. The treatment is rest and atropin.

Laceration of the Sphincter of the Iris (Radiating Laceration) is a common result of contusion. The tear is usually small, and is discovered only by the most careful examination. These small tears are the cause of many cases of traumatic mydriasis. Large lacerations may extend to the

ciliary body. They are rarely met with. There is no particular treatment for this condition. Atropin makes the tear gape, and therefore should not be used.

Inversion of the Iris is a rare condition due to trauma, and has not been satisfactorily explained as to its mechanics. The iris is turned backward and is doubled upon the ciliary body. The condition may be partial or complete. There is often an accompanying dislocation of the lens, and with it hemorrhage into the aqueous and vitreous chambers. Where the condition is total, absolutely no iris is to be seen. The treatment is entirely symptomatic. After the eye has become quiet attention should be directed to the refraction.

Traumatic Irideremia.—Total loss of the iris, the other ocular structures remaining intact, is an extremely rare condition, of which two examples have been recorded by de Beck. Both cases sustained blows producing rupture of the cornea and extrusion of the iris without injury to the lens.

Prolapse of the Iris.—After accidental injury, or following a cataract extraction, the iris may protrude through, or be caught in, the lips of a corneal wound. The diagnosis of iris-prolapse is easily made by inspection.

TREATMENT.—If the prolapse follows an accident, the surgeon may either attempt to replace the prolapsed portion or excise it. The former method, which is condemned because of the danger of infecting the eye, is accomplished by gently pushing the iris back into place with a clean spatula, after the conjunctiva has been cocainized and flushed with a solution of mercuric bichlorid or other antiseptic. After replacement a miotic is to be used three times a day for two or three days. Prolapse of the iris occurring after a cataract extraction should be treated by excision (iridectomy) when the condition is observed early. If it is noticed after firm adhesions have formed, it should not be interfered with.

Foreign Bodies occasionally rest in contact with the iris after perforating the cornea. They may move with the movements of the eye or may remain imbedded in the stroma of the iris. They are necessarily of diversified character, including fragments of iron, steel, guncaps, pieces of glass, grains of powder, etc. A rare condition is the transference of a cilium into the iris. A piece of steel or stone may rest in contact with the iris for a long period before causing inflammatory symptoms, but pieces of copper, even if aseptic, cause inflammation by chemic action. The detection of foreign bodies in contact with the iris is not difficult, provided the anterior chamber is not filled with blood or exudate.

TREATMENT.—Foreign bodies which are freely movable are to be extracted through a broad corneal section. Often the substance will be carried out by a gush of aqueous humor; if not, it can be drawn out by a magnet or seized with forceps. If the body is lodged in the iris, it can be removed by an iridectomy. Particles of powder, when driven into the iris, should be permitted to remain and the eye should be treated with atropin. Siderosis may follow the prolonged lodgment of pieces of iron or steel in the iris.

MOTOR DISTURBANCES OF THE IRIS.

The movements of the iris are indicated by change in the size of the pupil. Permanent dilation *(mydriasis)* and contraction *(miosis)* have been mentioned in Chapter IV. *Hippus,* a clonic spasm of the sphincter pupillæ, characterized by constant changes in the size of the pupil, is found in some cases of hysteria, epilepsy, neurasthenia, cerebro-spinal sclerosis, disseminated sclerosis, and in the early stage of acute meningitis. It may be present in normal individuals.

Iridodonesis, tremulousness of the iris on movement of the globe, shows a lack of support, and, while not a functional motor disturbance of the iris, will be mentioned in this place. It is often seen after simple extraction of cataract. It exists in cases where the lens is present, but is shrunken or luxated; in cases of fluid vitreous; in persons with a high amount of myopia; in some nystagmatic eyes; and sometimes follows slight blows upon the eye. In the last-named cases, if the lens is not partially dislocated, there is probably a rupture of some of the fibres of the zonula, establishing a communication between the posterior and vitreous chambers. There is no treatment for this condition.

CHANGES IN THE ANTERIOR CHAMBER.

Depth.—The anterior chamber is deepened in the myopic and shallowed in the hypermetropic eye. Its depth in the normal eye varies, becoming less in accommodation owing to the forward movement of the anterior surface of the lens. It is lessened in infancy and in old age. Pathologically the chamber is shallowed in traumatic cataract as well as in a stage of ordinary senile cataract. In glaucoma, particularly in the acute inflammatory type of the disease, it is of less than normal depth. Intra-ocular tumors cause a shallowing. In some old cases of inflammation of the uveal tract, with detachment of the retina, there is a lessened secretion of aqueous humor and a shallowing of the chamber. In iritis with the formation of circular synechiæ the chamber becomes shallow.

Contents.—In iritis, iridocyclitis, and keratitis punctata the aqueous humor becomes turbid. In glaucoma it is turbid and contains a greater percentage of solids than under normal conditions. Hypopyon, a collection of leucocytes and fibrin, is often seen in ulcers of the cornea and in violent attacks of iritis and iridocyclitis.

Hyphemia, a collection of blood in the anterior chamber, is not infrequently seen after injuries, both accidental and operative. It occurs idiopathically in severe cases of iridocyclitis, in hemorrhagic glaucoma, in hemophilia, splenic leucocythemia, and in intra-ocular neoplasms. Dislocation of the lens into the anterior chamber may occur. Cilia are rarely carried into it through corneal wounds. The lodgment of such foreign bodies as pieces of iron, steel, copper, or stone often occurs. The parasites which have been found in this locality are cysticercus and filaria.

OPERATIONS ON THE IRIS.

Of the numerous operations which have been devised and performed upon the iris, few are at present considered of value. They are iridectomy, iridotomy, and iridocystectomy. Corelysis, or the tearing of posterior synechiæ, and iridodesis—the tying of a piece of iris outside a corneal

Fig. 271.—First step in the operation of iridectomy. (AUTHOR.)

wound—have justly been abandoned by reason of their producing sympathetic ophthalmitis.

Iridectomy.—This is an excision of a piece of the iris. It may be done for an optical or for a therapeutic purpose. In the former, a com-

Fig. 272.—Second step in the operation of iridectomy. (AUTHOR.)

paratively small piece of the iris is generally excised, while a therapeutic iridectomy requires the removal of about two-fifths of the membrane.

OPTICAL IRIDECTOMY.—This operation is indicated in the following conditions: (1) in obstruction or occlusion of the pupil, (2) in central corneal and lenticular opacities, and (3) in high degrees of keratoconus.

THERAPEUTIC IRIDECTOMY is indicated: (1) in glaucoma; (2) as preliminary to cataract extraction; (3) in prolapse of the iris; (4) to remove tumors, parasites, or foreign bodies from the iris; (5) in chronic iritis; (6) to hasten maturation of cataract; (7) in corneal fistula; (8) in adherent leucoma and partial staphyloma, and (9) in the treatment of certain corneal ulcers which threaten perforation.

The required instruments are speculum, fixation forceps, angular keratome, iris-forceps, scissors, and spatula. If the operation is to be done upon an aphakic eye, a blunt iris-hook will be needed. While an optical iridectomy may often be done under local anesthesia, a therapeutic iridectomy usually requires general anesthesia. In children, nervous individuals, and insane patients chloroform or ether should be used. If a miotic has been used in the treatment of the case, it should be withdrawn several hours before iridectomy is attempted. The author has seen iridodialysis from an attempt to make an iridectomy in a case in which the miotic was not

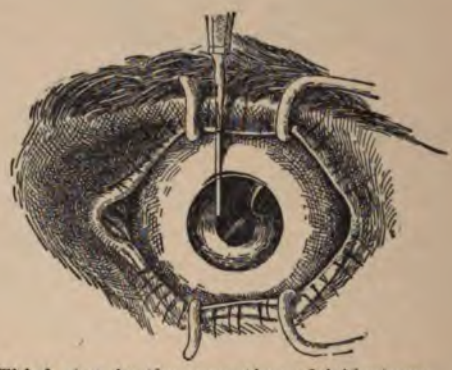


Fig. 273.—Third step in the operation of iridectomy. (AUTHOR.)

discontinued. The steps in an iridectomy are three: (1) opening of the anterior chamber; (2) the seizure, withdrawal, and excision of the iris; and (3) the toilet of the wound.

EXECUTION OF IRIDECTOMY.—The field of operation should be cleansed. The patient reclining on an operating-table with the speculum *in situ*, the surgeon seizes the globe with the fixation forceps, placed usually below, while the iridectomy is to be made above. If, however, the case is one of threatened corneal perforation from an ulcer or a recent wound, it will be inadvisable to subject the globe to such pressure as will obtain if the instruments are placed as described above. In such an event it will be wise for the surgeon to seize the ocular conjunctiva eight or ten millimetres behind the corneoscleral junction, while the keratome is passed in front (Fig. 274).

1. The Incision.—The keratome is to be passed through the cornea with a curved movement. At first the keratome is placed against the cornea at an angle of about 60 degrees; as soon as the point of the instrument is seen in the anterior chamber its direction is changed so that the point is

directed forward, thus avoiding injury to the iris, capsule, and lens. The keratome is gently pushed forward until an opening of sufficient size has been made. The instrument is then to be withdrawn slowly, to avoid the sudden escape of aqueous humor and rapid reduction of intra-ocular tension. "Perhaps the best idea of the action of the knife (keratome) is obtained by keeping in mind Arlt's direction, that it is to be employed as a curved needle" (Norris and Oliver).

2. Withdrawal and Cutting of the Iris.—Light, well-made, curved or angular iris-forceps, whose blades fit accurately, are introduced; the iris is seized near the pupillary margin and is gently drawn out of the wound. Undue traction on the forceps must be avoided. Cutting of the iris is then done with a pair of ordinary iris-scissors, or, as the author prefers, with the instrument of de Wecker.

3. Replacement of the Iris-angles is done by gentle strokes with the spatula.

In iridectomy for glaucoma the keratome is passed, not through the cornea, but through the sclera, one or two millimetres behind the cornea. The iris is withdrawn, cut at one end, then at the other, and finally at the base, care being taken to remove it far back to the periphery. The angles are then replaced as described above.

An iridectomy which is made preliminary to a cataract extraction will require the excision of only a small amount of tissue. Many surgeons remove only a minute piece adjacent to the pupillary border *(sphincterectomy)*.

The after-treatment of an eye subjected to iridectomy is usually uneventful. A piece of aseptic gauze is placed over both eyes, which are bandaged. The dressing is changed daily, and for a few days a drop or two of atropin solution will be instilled to prevent adhesions. If the iridectomy has been made for glaucoma, the atropin is used once only: *i.e.*, at the first dressing. One such application will prevent adhesions, and will not cause increase of tension.

Accidents during Iridectomy. — Among the accidents which may occur during an iridectomy are: (1) the passage of the keratome between the corneal layers; (2) spitting of the iris; (3) detachment (iridodialysis); (4) failure to excise the pupillary margin, thus leaving the sphincter intact; (5) injury to the anterior capsule of the lens; (6) rupture of the zonula; (7) dislocation of the lens, in consequence of sudden reduction of intra-ocular tension, into the groove between the root of the iris and the ciliary processes; (8) after making the incision the cornea collapses, indicating the presence of a firm cicatrix behind the iris and lens and making an iridectomy impossible; (9) the incision may be too small to permit the introduction of the iris-forceps.

Iridectomy in Case of Obliteration of the Anterior Chamber. —*Gayet's Method* is as follows: After fixing the eye with the double fixation forceps a minute incision is made into the periphery of the anterior

chamber with the ordinary scarificator, by sawing movements, enlarging the incision with blunt scissors. Dr. G. C. Harlan, of Philadelphia, has found this method useful in a case of chronic iritis with increased intra-ocular tension. The pupil was completely adherent at its margin, while the *iris bombé* lay directly against the cornea. It would have been impossible to make an incision with the keratome or von Graefe knife without involving the iris and possibly endangering the lens. He has found the narrow Weber canaliculus knife useful in enlarging the incision, its probe-point passing readily between the iris and cornea.

Deschamps's Method.—In aphakial eyes, with obliteration of the anterior chamber, the iris lies against the cornea, and the making of an iridectomy by the ordinary procedure or by Gayet's method will be practically impossible. In such cases Deschamps passes a cataract-knife at the corneal limbus through the iris and into the vitreous humor. The instrument is made to emerge at a point four or five millimetres away and the intervening tissues are cut. The iris is then seized with forceps from behind. It is

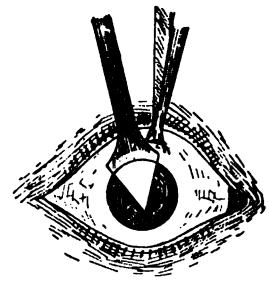


Fig. 274.—Iridectomy in threatened corneal perforation.

separated from the cornea, drawn out through the wound, and a piece is excised.

Transfixion of the Iris.—In cases of *iris bombé* an operation which gives excellent results is the transfixion procedure. A very narrow von Graefe cataract-knife is passed through the cornea and bulging iris and withdrawn. The cut establishes a communication between the anterior and posterior chambers. At the same time retraction of the distended membrane practically forms a new pupil.

Iridenkleisis.—This operation, which was devised by Eugene Smith, of Detroit, is employed for the relief of iridodialysis. A keratome is passed somewhat obliquely through the cornea at a point in front of the detached portion of the iris. The torn edge of the iris is seized by means of iris-forceps and a small piece of the iris is drawn into the corneal wound, where it is held fixed by the compression of the lips of the wound.

Iridotomy.—This operation, sometimes called iritomy, is an old procedure which was advocated by Woolhouse in 1711, first performed by Cheselden in 1728, and revived in recent years by de Wecker. It has undergone many vicissitudes. At the present day, however, it is performed

only on cases where iridectomy is impossible or dangerous. Although de Wecker sometimes performs this operation with the lens intact [(1) in zonular cataract, (2) central opacities of the cornea, (3) certain adherent leucomas, and (4) subluxation and luxation of the lens], among American and English surgeons its use is limited to cases in which, after removal of the lens, an adherent pupil exists. Formerly a broad needle or cataract-knife was passed into the anterior chamber and the iris (and tissues adherent

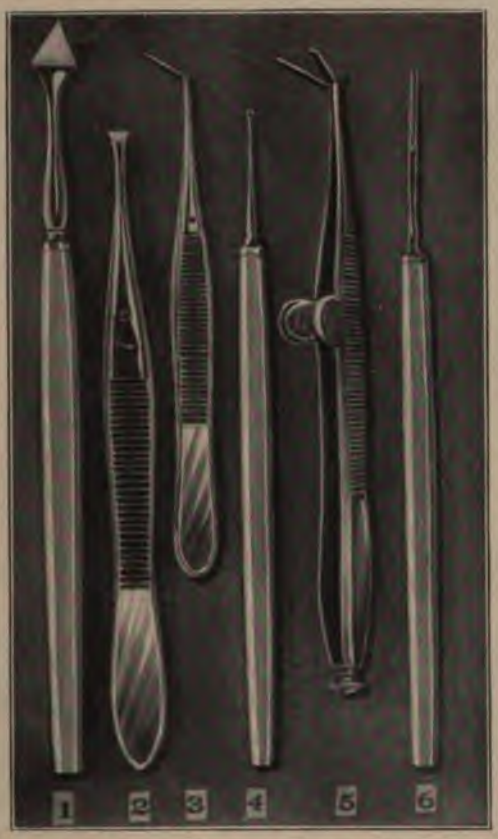


Fig. 275.—Instruments used in operations on the iris. (AUTHOR.)

1, Angular keratome. *2*, Fixation forceps. *3*, Angular iris-forceps. *4*, *Pince-ciseaux* of de Wecker. *5*, Spatula. *6*, Blunt hook. The speculum has been omitted.

to its posterior surface) was incised, the direction of the incision being across the iris-fibres. Contraction of the lips of the wound served to keep the artificial pupil open. The results were often unsatisfactory, leading surgeons to adopt the following operation:—

Iridocapsulotomy.—This operation is indicated in certain forms of secondary cataract. Its object is the creation of an artificial pupil without excision of the iris. The necessary instruments are speculum, fixation forceps, keratome, one narrow von Graefe knife, and the *pince-ciseaux* of

de Wecker, of which one blade is sharp and the other blunt. The speculum being in position and the eyeball fixed, the surgeon passes the keratome through the cornea at a point one millimetre from the corneal limbus. The exact location of the corneal incision will depend upon the direction in which the thickened iris and lens-capsule are drawn. If the condition demanding this operation has followed a cataract operation, in which the upper corneal section was made, the keratotomy for the operation of iridocapsulotomy will be placed at the temporal end of the horizontal diameter of the cornea. The keratome is quickly withdrawn and the *pince-ciseaux*, with

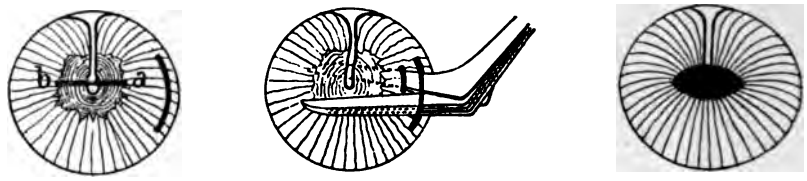


Fig. 276.—Iridocapsulotomy. (De Wecker.)

blades closed, is passed into the anterior chamber. The sharp blade is made to pierce the iris and lens-capsule, at a point two or three millimetres within the limbus. The instrument having been passed toward the nasal side, the blades are quickly closed, thus cutting the obstructing tissues (Fig. 276), which, if elastic, will retract and thus leave the desired pupillary opening.

There are several modifications of this operation. If the pupil is drawn far upward, or is concealed by the corneal cicatrix, the operation represented by Fig. 277 will be valuable. A keratome is passed through the cornea and also through the iris, making an iridal incision *(a-b)* three or four milli-

Fig. 277.—Iridocapsulotomy. (De Wecker.)

metres in length. At each extremity of this incision a cut *(a-c; b-d)* is made with the *pince-ciseaux*. If the tissues are elastic, a large pupillary opening will result. If the tissues do not retract, the procedure must be changed into an iridocystectomy.

Iridocystectomy (Irido-ectomy).—This is an excision of a part of the iris and thickened posterior capsule, and is indicated when, after a cataract operation or a traumatism, the pupil is closed by iridocyclitis or iridocapsulitis. The required instruments are a speculum, fixation forceps, Beer's cataract-knife or a keratome with a long narrow blade, Mathieu's capsule forceps, and ordinary iris-scissors or de Wecker's *pince-ciseaux*.

DE WECKER'S METHOD.—A keratome is passed through the cornea and iris. From each extremity of the iridal incision a cut is made with the *pince-ciseaux,* thus excising a triangular segment of the obstructing membranes. The incision of the cornea and iris may be made with the narrow cataract-knife of von Graefe. This is followed by an excision of a triangular segment of the iris and capsule.

KNAPP'S METHOD.—With the Beer knife the surgeon cuts through the lower part of the cornea (if the cataract extraction was made above) and pierces the iris and adherent capsule, making a wound three and one-half to four millimetres long. A blunt hook is used to draw the lower lip of the iris outside the wound. In some cases the hook fails and the capsule forceps of Mathieu will be necessary. A piece of the iris and adherent capsule is then cut off, the eye is bandaged, and atropin is used for a few days. Healing is usually uneventful. The visual results of this operation are generally excellent.

CHAPTER XI.

DISEASES OF THE CILIARY BODY.

THE ciliary body, placed between the iris in front and the chorioid behind, participates in the diseases of these structures. This portion of the uveal tract is subject to congenital defects, to neoplasms, to inflammations, and to injuries.

Paralysis and spasm of the ciliary muscle are discussed elsewhere (Chapter XX).

Fig. 278.—Coloboma of the iris and ciliary body. (BOCK.)

Coloboma of the Ciliary Body is a rare anomaly which occurs either in connection with coloboma of the iris and chorioid or alone. Heretofore coloboma of the ciliary body has been seen only in a downward direction; nevertheless an anatomic study of those rare cases of iridic and chorioidal colobomata occurring together, in which the fissure is atypically directed, would probably show a similar condition of the ciliary body. Anomalies of this body present many forms: the coloboma may be covered by a yellow mass consisting of an enormous hyperplasia of the non-pigmented cells of the pars ciliaris, which may project into the vitreous humor; the ciliary muscle may be unchanged, while the ciliary processes show a pronounced cleft; or the ciliary body may be entirely wanting in the region of an iris coloboma. It is evident that the condition can be recognized only when an accompanying coloboma of the iris extends to the periphery. There is no treatment for these conditions.

Tumors of the Ciliary Body.—Neoplasms originating in, and parasites transported to, the iris are certain in time to involve the ciliary body, which, however, may be the primary seat of a new growth or may be involved by

metastasis. With the exception of tubercle and gumma, primary tumors of this part of the eye are very rare. Such a growth will cause iridodialysis, increased intra-ocular tension, repeated hemorrhages into the anterior chamber, and loss of the eye. By focal illumination the tumor can sometimes be seen as a dark mass lying behind the iris. The diagnosis will be difficult if the eye is painful and hard, without the tumor presenting. The presence of nodules or bulging at the ciliary region will show that the neoplasm is about to break out. The following growths have been observed: sarcomata of various kinds, myomata and fibromyomata, carcinomata, and angiomata. The prognosis is serious, and the proper treatment is enucleation of the eye or exenteration of the orbit. Calcification and ossification are rarely found in this region. Parasites have been found in the ciliary body. In a few cases cysts have been found.

Fig. 279.—Leucosarcoma of the ciliary body and melanosarcoma of the iris. (KERSCHBAUMER.)

The lens is displaced and behind it an exudative membrane is seen. The optic nerve shows glaucomatous excavation.

Gumma of the Ciliary Body is a rare condition, appearing in from one to three and one-half years after the initial lesion of syphilis. The gummatous tumor is preceded by an attack of iritis. The vision is rapidly reduced, the eye showing great conjunctival and ciliary injection. The cornea becomes hazy, the anterior chamber deep, and hypopyon is present. A yellowish-red tumor is seen projecting from the angle of the anterior chamber, and at a spot corresponding to its site there is a bulging of the ciliary region. This is of a purple color. Tension becomes increased; there is great pain and considerable constitutional disturbance, as shown by the presence of anorexia, furred tongue, insomnia, and elevation of temperature. Other nodules appear, and these develop into ciliary staphylomata, presenting a bluish-black color, owing to the pigment showing through the thin sclera. Under proper treatment, which has been instituted and continued for several weeks, the eye becomes clear, the hypopyon disappears, and the tension gradually diminishes. Vision improves, but generally is not restored. The staphyloma may diminish in size, but some bulging always remains.

If the treatment is not efficacious, the eye becomes perforated, or atrophy of the globe may occur without perforation.

DIAGNOSIS.—This must rest upon the history and clinical signs as given above.

PROGNOSIS should always be guarded in these cases. If the patient retains the globe intact and possesses vision equal to the counting of fingers at a few feet, he should be congratulated. Stieren, however, has recorded a case of gumma of the ciliary body, with vision reduced to perception of light, which recovered vision ($^6/_8$) under enormous doses (100 to 200 grains, three times a day) of potassium iodid.

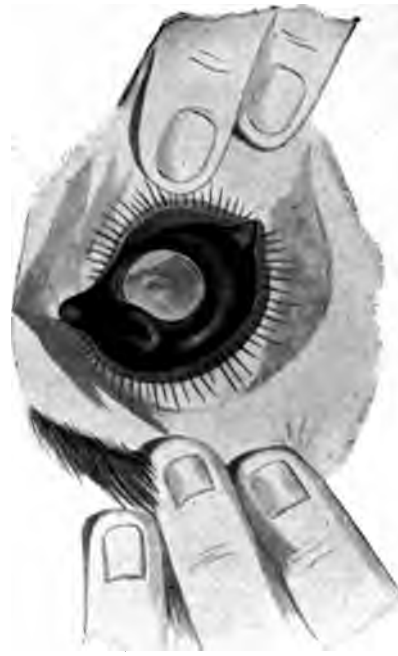


Fig. 280.—Gumma of the ciliary body. (NORRIS and OLIVER.)

TREATMENT.—Antisyphilitic remedies should be pushed vigorously. Locally atropin or scopolamin drops must be used, and the usual remedies are to be prescribed for the relief of pain.

INFLAMMATION OF THE CILIARY BODY.

Inflammation of the ciliary body, known as cyclitis, may be idiopathic or traumatic; acute or chronic; simple or complicated. As regards the pathologic process, it is divisible into serous, plastic, and purulent forms.

Serous Cyclitis (Descemetitis; Aquocapsulitis; Keratitis Punctata; Serous Iridocyclitis) is frequently described under the name of serous iritis, but properly comes under the head of this chapter, since Treacher Collins

has found that it depends on pathologic changes in the glands of the ciliary body. The disease presents the following

SYMPTOMS.—The corneoscleral region shows a delicate rose-colored injection. The anterior chamber is of normal or increased depth. The pupil is of normal size or slightly dilated. The tension is slightly increased at first, but diminishes later. The tendency to the formation of posterior synechiæ is not so marked as in iritis. The iris reacts slowly to light. Pain is rarely a prominent symptom. Exudation into the anterior chamber is moderate in amount, causing the aqueous and cornea to become cloudy. A deposit of opaque dots, often arranged in triangular form with the base downward, appears on the posterior elastic lamina of the cornea. The visual field may be contracted. A central scotoma may be present.

Serous cyclitis may exist alone, but generally it is found associated with chorioiditis, interstitial keratitis, or scleritis.

ETIOLOGY.—The disease occurs chiefly in ill-nourished, anemic young persons, particularly in women suffering from uterine diseases. According to Horner, it is more frequent in women than in men in the proportion of 10 to 3. Some cases are undoubtedly due to syphilis, either early or as a late manifestation, while others have been attributed to rheumatism, gout, eyestrain, or to trauma. A serious form of this affection is caused by sympathetic ophthalmitis.

PATHOLOGY.—The investigations of Nicati, in 1891, showed in rabbits the existence of glands in the ciliary body for the secretion of aqueous humor. Treacher Collins, working about the same time, demonstrated by bleached sections the existence of similar glands in the human eye. Serous cyclitis is a catarrhal inflammation of the ciliary glands. Hyperemia is followed by increased secretion and deepening of the anterior chamber. The aqueous humor becomes more albuminous than normally. A few leucocytes, together with pigmented epithelial cells thrown off from the surfaces of the glands, and shreds of fibrin are present. These solid elements drop to the bottom of the anterior chamber and form the dots of so-called "keratitis punctata." Leucocytes collect in the ligamentum pectinatum and hinder the egress of aqueous, thus causing increased tension. The turbidity of the aqueous humor causes the iris to lose its lustrous appearance. Severe cases may end in involvement of the vitreous humor with shrinking of the globe and development of cataract.

PROGNOSIS.—Except as a manifestation of sympathetic ophthalmitis, serous cyclitis offers a favorable prognosis. Adhesions do not often form between the iris and lens-capsule, but sometimes permanent opacities are left in the cornea and vitreous humor. The disease runs a slow course. When associated with marked fundus changes it may lead to blindness.

TREATMENT.—The internal use of mercury and potassium iodid is of value in these cases. Mercury used by inunction or hypodermically is an efficient remedy. If the patient is anemic, tonics are to be employed. In the cases due to gout and rheumatism, Turkish baths, diuretics, tonics,

and exercise, together with appropriate diet, are to be employed. As regards local treatment, the use of weak solutions of atropin is of value. If the tension is increased, paracentesis of the anterior chamber should be done, followed by the use of pilocarpin, arecolin, or weak eserin solutions. The presence of vitreous opacities will call for the vigorous use of mercury and diaphoretics internally. Use of the eyes for near work should be prohibited. As a rule, the patient should not be kept in a dark-room. The management of those cases which are due to sympathetic ophthalmitis will be described elsewhere (Chapter XVIII).

Plastic Cyclitis (Iridocyclitis Plastica) is the severest form of inflammation of the ciliary body. The symptoms are great pain, chemosis of the

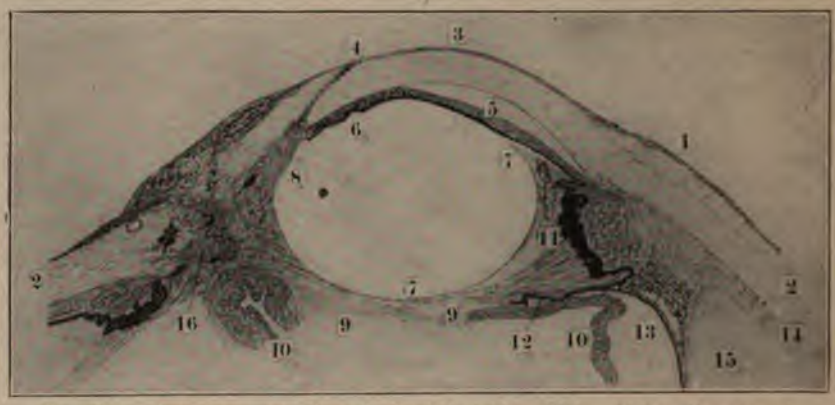


Fig. 281.—Anterior part of eyeball with iridocyclitis. (After POLLOCK.)

Section of an eye which received an extensive wound followed by hemorrhage, prolapse of the iris, and loss of vitreous. Iridocyclitis ensued, and the eye was enucleated thirty-nine days after the injury.

1, Conjunctiva. *2*, Sclerotic. *3*, Cornea. *4*, Cicatrix of the wound passing obliquely through the cornea, with pigment deposit. *5*, Stretched and atrophied iris adherent to the capsule of the lens. *6*, Pupillary margin of the iris which is adherent to the cornea (anterior synechia). *7, 7*, Capsule of the lens. *8*, Folded remains of part of the lens-capsule, imbedded in cicatricial tissue. *9, 9*, Newly formed connective-tissue membrane passing across the globe behind the lens. *10, 10*, Detached and folded retina. *11*, Ciliary body, dragged inward. *12*, Pigment layer of pars non-plicata of ciliary body. *13*, Pigmented cells in the chorioid. *14*, Chorioid. *15*, Blood-clot splitting the chorioid into two layers. *16*, Newly formed inflammatory membrane, showing fibres, round cells, pigment-vessels, and traces of the incarcerated and destroyed iris.

conjunctiva, and swelling of the lids. The eye is exceedingly tender to pressure over the ciliary region. There is great pericorneal injection, and exudation takes place into the pupil, behind the iris, and into the vitreous humor. Often the pupil is dilated by the contraction of plastic exudate deposited in the ciliary body. If the disease follows an attack of iritis, all the symptoms are accentuated. Early in cyclitis the tension is increased. The disease is accompanied at times by intense pain, vomiting, and fever. Photophobia and lacrimation are often present.

ETIOLOGY.—The most frequent cause of plastic cyclitis is traumatism (perforating wounds, cataract operations, etc.). It occurs idiopathically as a result of a similar process in the other eye (sympathetic ophthalmitis).

Stephenson—in view of the fact that numerous inflammatory diseases of the iris and chorioid result from depraved blood-conditions, which, in turn, are attributed to microbic causes—regards the excretion of microbes or their products by the ciliary body as the cause of all cases of endogenous iridocyclitis.

PATHOLOGY.—The first step in the morbid process is hyperemia with transudation of leucocytes. The characteristic feature of the disease is the fibrinous exudate, which appears on the inner surface of the ciliary body, on the zonula of Zinn, in the posterior chamber, and in the vitreous humor. The inflammatory process may end in resolution, the exudate being absorbed, or it may become organized. Vessels from the ciliary body or from the peripheral parts of the retina grow into it. The iris becomes adherent to the lens, thus obliterating the posterior chamber; and contraction of the newly formed membranes, with detachment of the retina and ciliary body, takes place. The nutrition of the lens being interfered with, cataract results. The eye finally becomes shrunken and soft: a condition known as *atrophy of the eyeball.*

PROGNOSIS.—This form of cyclitis is rarely improved by treatment.

TREATMENT.—The treatment is that of a severe case of iritis with the addition of large doses of mercury and potassium iodid, together with diaphoretics. If the eye becomes blind and shrunken and is painful to the touch, it should be enucleated.

Purulent Cyclitis.—This disease is caused by infected wounds and acute infectious diseases. It runs a sharp course. Hyperemia, round-cell infiltration, and the presence of pus in the anterior chamber are prominent pathologic features. The symptoms are all accentuated. If the disease is due to a wound, the eye is lost by panophthalmitis. Purulent cyclitis sometimes appears idiopathically in the course of influenza, small-pox, and scarlatina.

PROGNOSIS.—This is unfavorable.

TREATMENT.—This will consist of hypodermic injections of morphin to relieve pain and the local use of atropin, hot compresses, and bichlorid solution. Inunctions of mercury and subconjunctival injections of the same drug may be employed. Most cases of purulent cyclitis end in phthisis bulbi and require enucleation.

Injuries of the Ciliary Body are among the most dangerous traumatisms of the eye, and are particularly prone to cause sympathetic ophthalmitis. The iris and lens are almost invariably injured at the same time. If the instrument producing the trauma remains within the globe, the case becomes much complicated, and comes under the head of retained foreign body. If the instrument has been withdrawn, and has not carried infection into the eye, the case may do well, or the eye may be lost by a slow iridocyclitis. Careful inspection is necessary in all eye injuries. In the management of these cases cleanliness should be absolutely observed by the surgeon. If the wound is of considerable extent and involves the sclera, it can be

treated by snipping off any protruding bead of vitreous humor and by suturing the sclera. Whenever possible to do so, the scleral wound should be covered by a sliding conjunctival flap. It is only in those extensive lacerations where there is no hope of vision that an immediate enucleation should be made. Under any other circumstances the patient is to be given the benefit of skilled advice. In these cases the general practitioner will not be doing his duty unless he observes absolute cleanliness and refers the case to a specialist.

CHAPTER XII.

DISEASES OF THE CHORIOID.

THE chorioid coat is part of the uveal tract and is characterized by its vascular and pigmentary supply. It is subject to congenital anomalies, tumors, inflammations, and injuries.

CONGENITAL ANOMALIES.

Coloboma of the Chorioid and Retina may be considered together, since for practical purposes there can be no coloboma of one tunic without

Fig. 282.—Coloboma of the chorioid. (BENSON.)

the other being absent, although microscopic examination sometimes demonstrates the presence of the retina in cases of chorioidal colobomata. Eyes in which portions of the chorioid and retina are wanting are often hypermetropic, nystagmatic, strabismic, and amblyopic. The defect is usually situated in the lower part of the fundus. Generally the iris presents a coloboma also, but cases are on record in which coloboma of the chorioid was the only malformation. With the ophthalmoscope the coloboma is seen as a whitish, pearly patch, of round or ovoid shape, often with a pigmented border, presenting a few retinal vessels in its area. The sclera may bulge at the site of the coloboma and resemble a cyst. Microphthalmos and imperfect development of the nervous system and of the skull are not infrequent accompaniments of chorioidal colobomata. The surface of the

coloboma can be seen to be deeper than that of the surrounding retina. Similar white depressions are sometimes seen in the macular region and are called macular colobomata. Of these, about 40 cases are recorded, of which 5 were bilateral. A coloboma of the macula usually presents a white or yellowish area of a round or oval shape, whose border often shows pigmentation. It is traversed by retinal vessels. In rare instances chorioidal vessels are present resembling vascular convolutions, and this condition has led Lindsay Johnson to regard macular colobomata as the atrophied remains of nevoid growths in the chorioid. Most cases of macular coloboma show myopia. Visual acuity may range from high amblyopia to normal vision. The field of vision shows a defect, scotomata of various forms being found in different cases. In a nystagmatic eye, to differentiate between chorioidal atrophy and coloboma may be difficult. Aside from the correction of the refraction, there is no treatment for coloboma.

Fig. 283.—Coloboma of the macula, with islets of pigment and an atrophic crescent of the optic disc. (SILEX.)

Albinism (Leucosis).—Congenital absence or insufficiency of pigment in the uveal tract causes the iris to look pinkish or yellowish, from reflection of light from the iridal and chorioidal vessels. The chorioidal vessels are visible ophthalmoscopically. Nystagmus, strabismus, errors of refraction, and reduction in visual acuity are frequent accompaniments of albinism. Treatment is limited to the correction of errors of refraction and to the shading of the eyes with dark glasses.

TUBERCULOSIS OF THE CHORIOID.

Tubercles of the Chorioid (Fig. 2, Plate XIII) may be in the form of miliary tubercles accompanying general acute tuberculosis, or as large masses in chronic tuberculosis. In *miliary tubercle* a few tubercles (one to ten) are found in the macular region or are scattered over the fundus. Each tubercle usually measures from one-third to two-thirds the diameter of the optic disc,

but may be larger. It appears as an oval, round, or reniform spot of a pale-rose, grayish-yellow, fawn, or paper-white color. Its edges appear "moth-eaten," and are marked by a narrow zone of retinal pigment. Its surface may present specks of pigment, dots, or pinkish stripes. They resemble the spots seen in disseminated chorioiditis. When multiple, the common number is two or three. The tubercles in this form of the disease appear a few days or weeks before death; hence, they cannot be mistaken for the elevations of disseminated chorioiditis. They begin in the deeper layers of the chorioid, growing from the adventitia of the vessels. They do not affect the vision. In many cases they can be demonstrated by microscopic section in eyes which present no changes to the ophthalmoscope. In probably 80 per cent. of cases of miliary tuberculosis their presence can be demonstrated post-mortem. According to Carpenter and Stephenson, they are found ophthalmoscopically in 50 per cent. of cases of acute miliary tuberculosis and tuberculous meningitis, and in 10 per cent. of cases of chronic (surgical) tuberculosis. The presence of tubercle bacilli cannot always be demonstrated in the cases. The larger tubercles show giant cells with a reticulum of fibres, epithelioid cells, small-cell infiltration, and caseation. The smaller growths are collections of lymphoid cells situated between the vessels. Since this disease appears very late in an incurable malady, treatment is valueless. The presence of tubercle in the chorioid may assist the physician in making a diagnosis between acute miliary tuberculosis, typhoid fever, and meningitis. While of serious import, in rare instances they may disappear or become quiescent. In chronic tuberculosis the chorioidal lesion may be single, multiple, disseminated, or diffused. The treatment is limited to hygienic and constitutional measures.

A Large Tubercular Mass often presents the clinical picture of a sarcoma of the chorioid. It is probably always secondary to tuberculosis located elsewhere, although the latter may defy localization. The disease is usually unilateral. In rare cases the patient may early show a destructive inflammation of the globe with perforation, or the disease may cause no symptoms and may be accidentally found by ophthalmoscopic examination. In a few cases glaucoma appears. Since the disease occurs chiefly in children and young subjects, it may be mistaken for neuroepithelioma of the retina. There is much variety in the ophthalmoscopic appearances of this disease. The tubercular mass usually appears as a solitary white or grayish elevation, springing from the central part of the fundus. It is to be distinguished from leucosarcoma of the chorioid, neuroepithelioma of the retina, and subretinal cysticercus. The absence of vessels serves to exclude the first and second conditions, while its immobility and solidity will distinguish it from cysticercus.

Since the tubercular tumor is secondary to tuberculosis located elsewhere, the removal of the eye is not demanded at an early stage, provided the diagnosis is clear. If it is in doubt, the eye should be removed and subjected to microscopic examination. Cases of tubercular tumor which

are progressive and tend to perforate should be treated by enucleation. The general treatment of the patient is of great importance in these cases. After a successful operation the patient may die from tuberculosis of the meninges, lungs, or abdominal viscera.

TUMORS OF THE CHORIOID.

Carcinoma of the Chorioid is of rare occurrence, Oatman having collected only thirty cases. The disease is always metastatic. The rarity of metastatic growths in the eye is accounted for by anatomic conditions: the small size of the ophthalmic artery and the fact that it is given off from the internal carotid at an angle of ninety degrees. While metastasis should be more common in the left eye than in the right, owing to the difference in the carotids, statistics do not show this to be true. The left carotid should receive emboli more easily than the right, inasmuch as it rises from the aorta directly. Metastatic growths occur most often at the posterior pole of the eye, owing to the great calibre of the short ciliary arteries. Of 28

Fig. 284.—Metastatic carcinoma of the chorioid.

cases in which mention is made of its location, the primary growth was situated in the breast in 20 cases; in the lungs in 3, in the liver in 2, in the stomach and liver in 1, in the suprarenal capsule in 1, and in the thyroid gland in 1. In one-third of the cases the disease was bilateral. The ages of the patients ranged from thirty to fifty-eight years. The disease is twice as frequent in females as in males. Vision is destroyed within a few weeks after the appearance of ocular symptoms. The average duration of life is about six months.

The disease appears ophthalmoscopically as a "flat, oval deposit or tumor, on the temporal side of the nerve, involving the macula with a central elevation of + 3 D., its edges gradually fading off into the surrounding fundus. Its color is a dirty-yellow, with scattered pigment spots" (Mitvalski). No vessels are apparent in the growth. The retina becomes detached at an early period. The tension of the affected eye may be normal, increased, or reduced. Enucleation may be necessary to relieve pain.

Other Tumors of the Chorioid.—Aside from the occurrence of carcinoma and the solitary form of tubercle, which have been considered, the chorioid is the seat of sarcomata, myomata, cysts, and nevi. Sarcomas

occur either primarily or by metastasis. They belong to the middle period of life, being rarely seen before the thirty-fifth year. Some are white (leucosarcomata) while others are black (melanosarcomata). The latter derive their color either from the presence of melanin or from the pigment of the blood. Their tendency is to grow out of the globe in three directions: (1) along the course of the venæ vorticosæ, (2) along the optic nerve to the brain, and (3) to the corneoscleral junction. They may perforate the sclera in other places. Their microscopic appearance is variable, and more than one type of structure may be found in the same growth. The small round-cell and spindle-cell tumors are common. Angiosarcoma, telangiectatic sarcoma, adenoma, perithelioma, and enchondroma are very rare chorioidal growths.

Sarcoma of the Chorioid.—This disease (Fig. 1, Plate XIII) is found in the proportion of about 1 to 3000 ophthalmic patients. In the vast majority

Fig. 285.—Fungating sarcoma of the chorioid. (AUTHOR.)

of cases the growth is primary. However, the author has met with one case of chorioidal sarcoma sequent to a sarcoma of the leg. In another metastatic case, which was observed by Meigs and de Schweinitz, the primary growth was located in the mediastinum. Doubtless metastatic sarcoma of the chorioid occurs more frequently than is supposed, since in many cases of sarcomatosis the eyes are not examined. The primary intra-ocular growth is usually single and unilateral, and generally is in the form of a projecting knob, growing toward the centre of the eye, and presenting a neck and base. Sarcoma may appear as early as the fifteenth or as late as the eighty-fourth year, although it is most frequent between the age of forty and sixty. The disease is said to be more common in males than in females (Fuchs, A. Hill Griffith, Lawson); but Kerschbaumer states that sex is without influence. After removal of the eye the disease often becomes metastatic, invading any organ or tissue, not excepting the bones. The brain is rarely involved by extension along the optic nerve. A rare complication is pig-

mentation of the skin, either in the form of a diffuse dark color, suggestive of argyrosis, or in the form of small, discrete round spots, two to four millimetres in diameter, which may be too numerous to be counted. In some cases of melanosarcoma of the chorioid melanin is found in the urine.

SYMPTOMS.—These vary much in different stages of the disease:—

1. In the first stage a detachment of the retina is seen at the site of the tumor. The mass appears of a purple-red or slate color according to its pigmentation; if deeply pigmented, it may appear black. Blood-vessels are seen coursing over it. There is no pain, and the tension is normal. The defect in the visual field may take an hemianopic shape or be quite irregular. Vision in the first stage may be only slightly or considerably reduced, according to the proximity of the growth to the macula. The duration of the first stage is from one to two years.

2. In the second stage there is increase of tension, while the external appearance of the eye resembles that found in inflammatory glaucoma: the anterior chamber is shallow, the pupil dilated, the cornea cloudy, and dilated episcleral vessels are visible. If the media are clear, a detached retina is seen. Later the lens becomes opaque and the clinical picture is that of absolute glaucoma. Pain now appears and makes the glaucomatous picture so striking that experienced diagnosticians have been deceived.

3. In the third stage the tumor escapes from the globe, the symptoms varying according to the place of exit. If in front, dark nodules are seen about the corneoscleral region. The area of impending perforation bulges and presents a rich development of blood-vessels, together with a black or bluish discoloration (Fig. 4, Plate XII). If behind, the existence of perforation cannot be told until the growth produces exophthalmos. Pain is relieved by the perforation of the globe. The growth of the sarcoma then proceeds with startling rapidity. The tumor fills the orbit and may cause a projection as large as two fists. The exposed parts of the mass ulcerate and bleed; the deeper portions may grow into the brain.

4. In the fourth stage, metastatic nodules form in the internal organs, the liver, stomach, and mesenteric glands being favorite sites. The patient dies by exhaustion. The first and second stages may occupy several years; the third and fourth stages are measured by months.

DIAGNOSIS.—In making a diagnosis of sarcoma of the chorioid the following conditions must be reckoned with:—

1. Detachment of the retina and chorioid.
2. Neuroepithelioma of the retina.
3. Solitary tubercle of the chorioid.
4. Intra-ocular cysticercus.
5. Glaucoma.
6. Spontaneous rupture of the eyeball.
7. Iridocyclitis.
8. Syphilis with chorioidal exudation.

1. *(a) Retinal Detachment* caused by sarcoma of the chorioid differs from the simple variety. In the latter the retina, early in the history of the case, is transparent, owing to the absence of the color given to it by the chorioidal pigment. Spontaneous retinal detachment is preceded by

PLATE XIII.

Diseases of the Choriod.

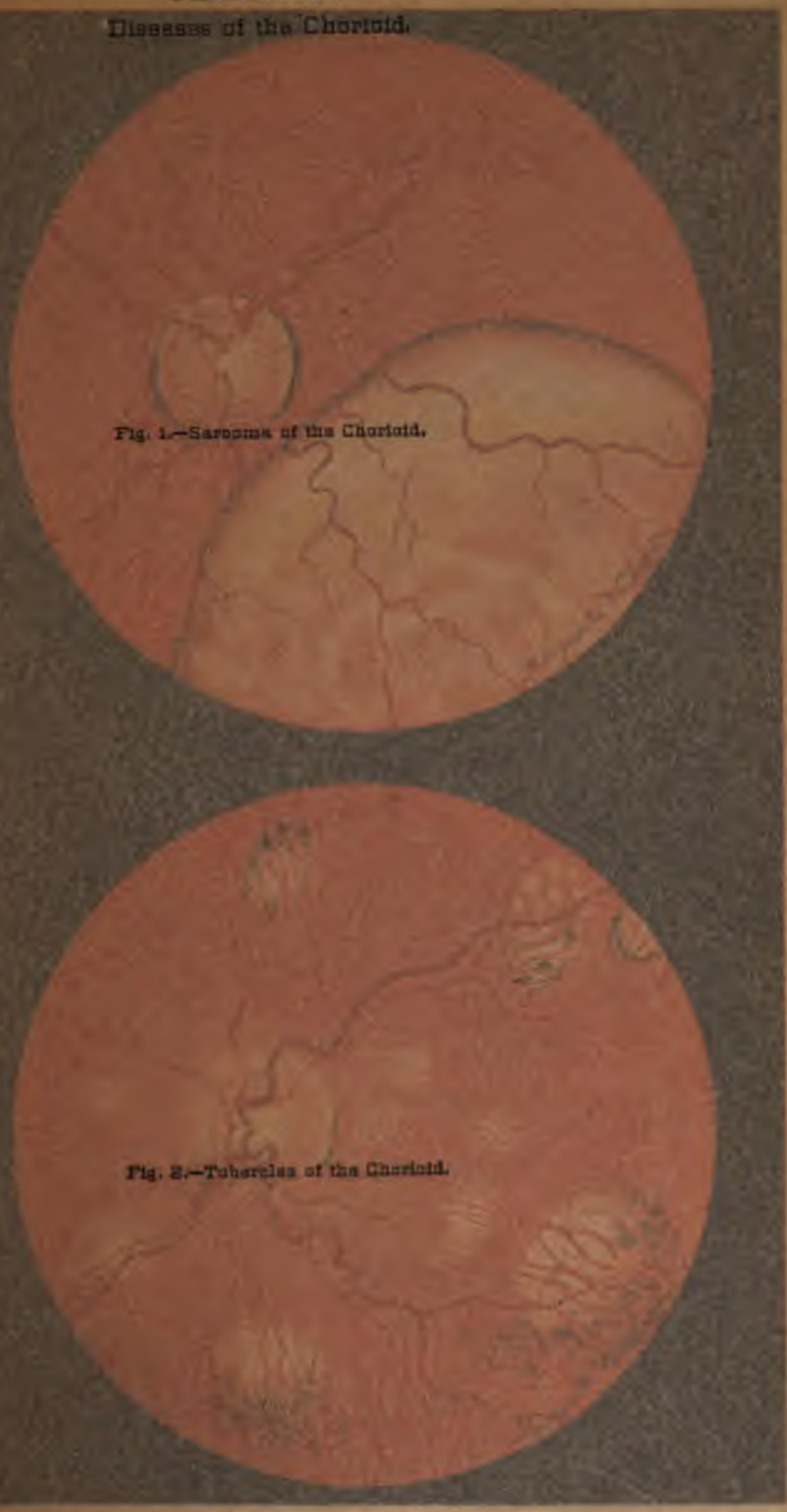
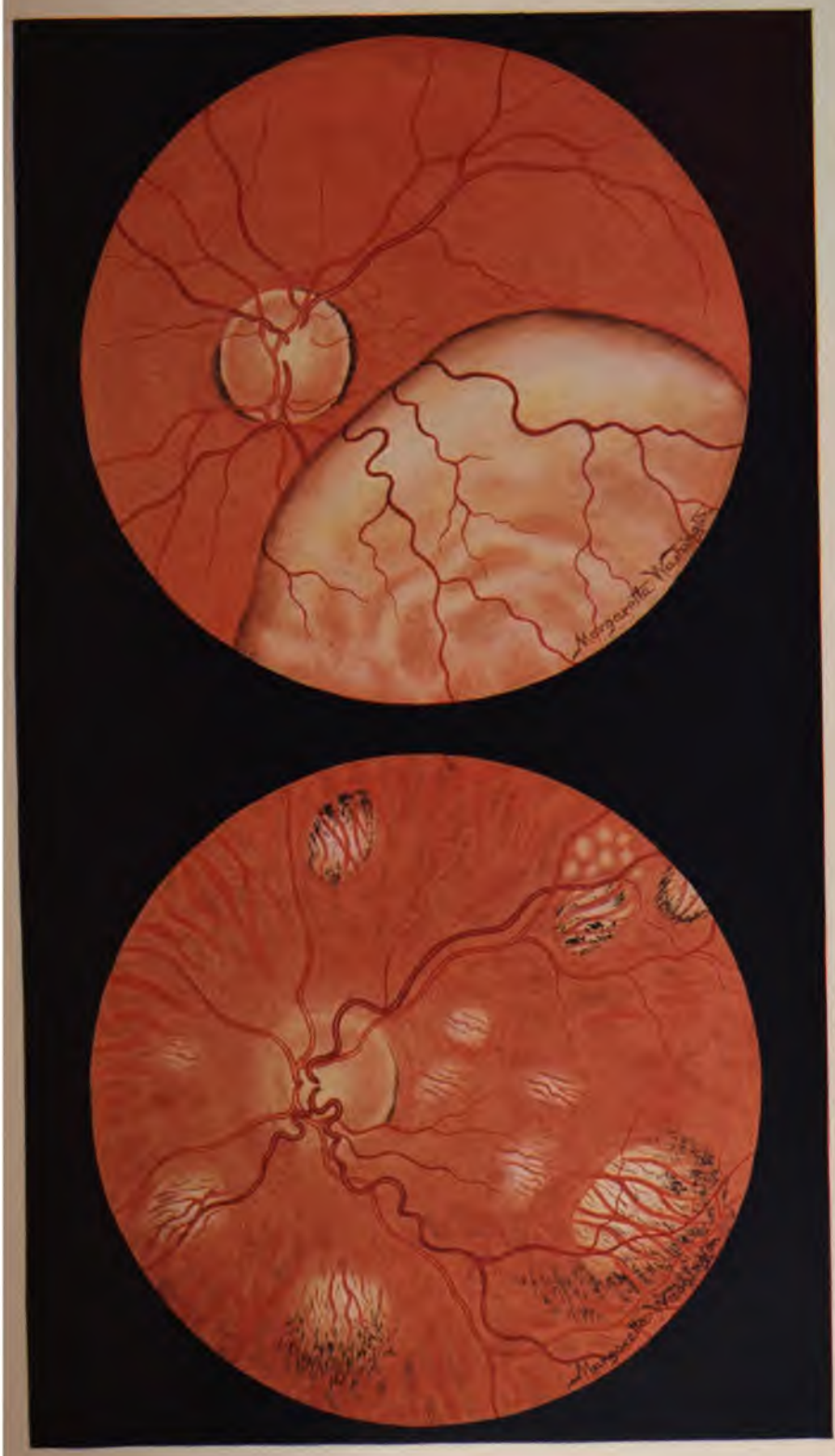


Fig. 1.—Sarcoma of the Choriod.

Fig. 2.—Tubercles of the Choriod.

PLATE 13



scæ volitantes. It appears suddenly, and is accompanied by vitreous cities, reduced tension, and signs of chorioiditis, and often occurs in s which are highly myopic. The condition of the other eye as regards action, the state of the fundus, and the history of the case are of some e. Holden says that the simple form of retinal detachment extends to ora serrata, while in tumor the detachment is longer delayed, particularly n the tumor is in the ciliary body or in the posterior pole of the eye. The chment in the simple form tends to produce folds and show undulations novement of the eye, while in tumor the folds are generally absent and mass does not undulate. However, cases occur in which serum col- s between a sarcoma and the retina, thus simulating simple detach- t and making the diagnosis difficult or impossible. In some of these s, where the retina is not thickened and the sarcoma is rich in vessels, tumor may be recognized by the presence of vessels which are neither rioidal nor retinal. As a rule, a large detachment of the retina, pressing inst the lens, will be due to an underlying sarcoma. Tension is a valuable gnostic point: in retinal detachment due to a serous collection it is ierally decreased; in chorioidal sarcoma it is usually normal at first l becomes increased later. A dilation of the anterior ciliary veins, when alized to one part of the globe, indicates sarcoma, the growth interfering h the return of the blood through one of the venæ vorticosæ. In melano- coma the urine sometimes contains melanin, and turns black on the dition of perchlorid of iron.

Bellarminoff's device may assist in making a diagnosis. The eye being esthetized, a piece of moistened plane glass is pressed against the cornea, us causing it to become flat and eliminating its refraction. By using rong illumination the opaque sarcoma may sometimes be recognized be- ath the detached retina. Tubular devices for trans-scleral illumination ay be of value. Light is concentrated on the sclera while the surgeon notes he fundus reflex. When the apparatus is placed opposite an intra-ocular umor, the fundus reflex becomes dimmer.

In case of doubt, the diagnosis resting between sarcoma and retinal detachment, Hirschberg proposed to puncture the eye. If a sarcoma is present, blood will be drawn; if the case is one of detachment, only serous fluid will be obtained. Hirschberg, however, has abandoned this procedure. It is uncertain, and, furthermore, it may furnish a path by which extra-ocular extension may occur. In any instance of doubtful diagnosis, the vision being markedly reduced or entirely lost, an enucleation will be justified on the ground that it is better to remove a dozen useless eyes, which are blind from retinal detachment, than to leave a case of chorioidal sarcoma till the third stage is reached.

(b) Detachment of the Chorioid presents even more problems in differential diagnosis. It forms a circumscribed projection, standing out prominently upon the fundus like a sarcoma. The foldings, present in simple retinal detachment, are absent in detachment of the chorioid. Usu-

ally in chorioidal detachment there is hemorrhage and chorioidal vesse can be recognized beneath the retinal vessels. Fortunately the conditio is extremely rare. Transient detachment of the chorioid, following catara extraction, has been observed by Fuchs (see Chapter XIII).

2. Neuroepithelioma of the Retina.—Here the age of the patient of importance. Neuroepithelioma is found only in children. While sa coma of the chorioid may occur in childhood, such cases are exceptional. the media are clear, a diagnosis between neuroepithelioma and sarcoma usually not difficult. The former presents a yellowish-white tumor (Fig. Plate XII). A sarcoma, if pigmented, looks much different. In a lat stage, when glaucomatous symptoms arise, diagnosis will be difficult.

3. Solitary Tubercle of the Chorioid may be mistaken for leucosarcom Tubercle, however, does not show vessels, and occurs, for the most part, young tubercular subjects.

4. Intra-ocular Cysticercus is of such rare occurrence in this count as to demand no consideration. In the early stages, before the advent inflammatory symptoms, the picture of cysticercus is characteristic a can hardly be mistaken for sarcoma.

5. Glaucoma. — The differential diagnosis between sarcoma of th chorioid and acute inflammatory glaucoma which has been untreated ma be impossible. The author once made an iridectomy for the relief of pa for what was diagnosticated as glaucoma absolutum. Improvement w but temporary; the pain returned and in a few weeks an enucleatio showed the case to be sarcoma of the chorioid. In such a case the med are opaque, the eye is hard, the episcleral vessels are dilated, and the pain severe. It will rarely happen in glaucoma that one eye will be entire blind and the other eye normal.

6. Spontaneous Rupture of the Eyeball, of which the author h observed one case, and Gilfillan and Millikin have recorded others, presen a picture identical with that found in the third stage of sarcoma of th chorioid. The history of the case, and the excessive rarity of spontaneo rupture, will serve to clear the diagnosis.

7. Iridocyclitis.—Here the chief difficulty arises because of cloudi of the media, occlusion of the pupil, and opacification of the lens. It so times occurs that, in the course of its growth, a chorioidal sarcoma will ca chorioiditis and iridocyclitis of such intensity as to lead to phthisis b In a case of idiopathic iridocyclitis both eyes are frequently affected. sion is usually reduced, while in sarcoma tension is usually increased the period when the media are clouded. In traumatic iridocyclitis th tory of the case will clear the diagnosis.

8. Syphilis.—Post has recorded a case of sarcoma, occurring myopic syphilitic subject. There were floating opacities in the vi humor and a projecting mass was seen between the macula and the nerve head. No improvement followed antisyphilitic remedies. T was enucleated and was found to contain a leucosarcoma of the spin

type. In this connection it may be stated that Silcock has reported a case of gumma of the chorioid.

PATHOLOGY.—Sarcoma of the chorioid usually is a firm tumor, but is sometimes gelatinous and may undergo fatty, myxomatous, osseous, or cartilaginous degeneration. Histologically it consists either of round or spindle cells or a mixture of both. It is usually of the small-cell variety. It arises either from the outer or middle layers of the chorioid, and its tendency is to grow inward toward the vitreous. Rarely the tumor is flat; its common form is spheroidal as long as its chorioidal covering is intact. Later it breaks through the lamina vitrea and assumes a spheric shape. In its growth it detaches the retina throughout a large area, but at the apex the tumor and retina are intimately attached. Following the detachment comes a stage of inflammation, in which either glaucoma or iridocyclitis is set up, the latter leading to atrophy of the eyeball. Occurring under these circumstances, atrophy of the globe is supposed to be due to iridochorioiditis caused by degeneration in the neoplasm, and by microörganisms (Leber, Krahnstöfer, Evetsky). Kipp has recorded a case of chorioidal sarcoma followed by atrophia bulbi. Twenty years later perforation of the globe and rapid growth of the neoplasm occurred. The stage of extension follows, the tumor spreading along the optic and ciliary nerves, or along the course of vessels entering the globe. Nodules form in the orbit and thus exophthalmos is produced. Metastases form in distant organs by embolism, the blood-current detaching and carrying the cells to other parts. While local recurrences are unusual after cases operated upon at an early stage, metastases sometimes occur long after the removal of the eye. Sarcomas of the chorioid belong principally to the pigmented tumors (melanosarcomata). The leucosarcomata are rare.

PROGNOSIS in chorioidal sarcoma is always grave. If the tumor-mass has reached the third stage, it is generally absolutely bad. An early diagnosis and thorough operation made in the first or second stage will save about 40 per cent. of these cases. The danger of local return under such circumstances is small, but the liability to the development of metastases is great. These usually appear within six months after enucleation. If the patient remains well for four years after operation, the immunity is probable, but is not assured. In untreated cases the duration of life is said to be about five years. The prognosis is more favorable in spindle-cell sarcoma than in the round-cell variety.

TREATMENT.—Only surgical procedures are of value in chorioidal sarcoma. If the tumor has not passed beyond the second stage, an enucleation, with complete resection of the orbital part of the optic nerve, its membranes, and the surrounding tissue at the apex of the orbit, will be sufficient. If there is doubt about the second stage being past, or if the tumor has undoubtedly extended beyond the globe, a complete removal of the orbital contents must be effected. This can be done rapidly and efficiently, a scalpel, scissors, and forceps being the only instruments necessary. The

author believes that, with the modern methods of operating, the use of caustics in the orbit should be eschewed. In operating on cases in the third stage the aim of the surgeon is more to relieve pain and destroy fetor than to save life. Here the hypodermic injection of the toxins of erysipelas and of bacillus prodigiosus or exposure to the x-ray may be tried. In the fourth stage treatment is useless except to relieve pain.

Myoma of the Chorioid.—Although the interior of the eye is well supplied with material for the growth of muscular tumors, few cases of ocular myomata are on record. This form of tumor has been found several times in the chorioid. In the case reported by Guiata, of Siena, the diagnosis rested between a subretinal cysticercus and chorioidal neoplasm. The case was that of a man, aged 20 years, whose vision had been failing for months. The ophthalmoscope showed a rounded elevation. Repeated examinations failing to show movement in the mass, the diagnosis of chorioidal tumor was ventured, and an enucleation was performed. The tumor measured eight by five millimetres, and extended from the ora serrata to the equator. Microscopic examination showed it to be a myoma of the chorioid.

Ossification of the Chorioid is of very rare occurrence. Ossification and calcification in an intra-ocular exudate are not unusual conditions (see page 422).

INFLAMMATION OF THE CHORIOID.

The term chorioiditis is a broad one, and is applied not only to cases in which the inflammation of the chorioid is actually in progress, but also to the lesions which exist long after the subsidence of the inflammatory process. A diagnosis of chorioiditis with the ophthalmoscope is possible only after the pathologic process has involved the retinal pigment-cells; hence, the condition is a chorioidoretinitis. From a clinical standpoint, cases in which the pathologic process begins in the chorioid are classed as chorioiditis, which is divisible into (1) exudative and (2) suppurative forms. In the former the eye is of normal appearance externally, and the diagnosis can be made only by ophthalmoscopy; in the suppurative form the iris and ciliary body are involved, and in violent cases the condition known as panophthalmitis ensues. Serous chorioiditis, formerly held to be the cause of glaucoma and of detachment of the retina, does not occur.

Hyperemia of the Chorioid exists, but cannot be diagnosticated as a distinct condition, since its only ophthalmoscopic sign is increased vascularity of the optic disc, whose vessels anastomose with those of the chorioid. Hyperemia is attributed to eyestrain from uncorrected or improperly corrected errors of refraction. In such cases the hyperemia of the nerve-head disappears under treatment by atropin, rest, and colored glasses, or after the use of the proper correcting glass.

Exudative Chorioiditis (Plastic Chorioiditis).—Chorioiditis may be localized or disseminated. There are no subjective symptoms peculiar to the disease. Hence the diagnosis must rest on the ophthalmoscopic findings.

SYMPTOMS.—While there are no subjective signs characteristic of inflammation of the chorioid, it is the rule that the patient complains of reduced visual acuity, the presence of specks or a mist before the eyes, and a feeling of heaviness in and about the eyes. Frequently there are subjective sensations of light (photopsiæ) and distortion of images (metamorphopsia), due, respectively, to irritation and to a lifting up of the retina by the chorioidal exudation. Vision may be slightly or greatly reduced or even abolished, according to the proximity of the inflammation to the macula or to the dissemination of the process. Scotomata are frequently found. These subjective symptoms will suggest the necessity of an ophthalmoscopic examination.

The changes found in the fundus will depend upon the stage of the disease. In the early stage the alterations are slight; in the height of the exudative process they are more marked; and in the period of atrophy they are most striking. In the early stage there are found edema of the retina with the presence of minute, rounded, discrete, yellowish-white elevations situated beneath the retinal vessels. The elevations may be most numerous at the periphery; in fact, it is usually stated that disseminated chorioiditis begins at the periphery, but there are many exceptions, the macula often being the area first showing the disease. Simultaneously with the pouring out of the exudation into the chorioid there occurs a depigmentation of the retina over the involved area. Probably in all except the eyes of blondes this pigment destruction is necessary before the chorioidal exudate can become visible. Involvement of the retina is indicated by the presence of snowy-white or bluish-white areas; and when the retinal cloud is absorbed the chorioidal stroma is exposed to view. This change can be appreciated by comparing Fig. 286 with Fig. 287, from Liebreich's "Atlas," showing the same fundus with an interval of ten months. Hemorrhage into the chorioid and hyperemia and swelling of the nerve-head are not common accompaniments of the early stage of chorioiditis. The process may stop here, the exudate undergoing absorption and the disease leaving few traces; but, as a rule, such a favorable result does not occur. The process goes onward, or, if checked, a fresh attack of chorioiditis ensues. Pigment is deposited over and around the exudation; or, as often occurs in the form of chorioiditis accompanying myopia, the chorioid undergoes extensive atrophy with only slight pigment deposition. In the stage of atrophy chorioiditis presents a striking picture, the denuded sclera being visible. If the process has become arrested while the exudation spots were small and discrete, in the atrophic stage the fundus will show small, white, circular areas, each of which presents a pigment border. Frost states that this form of the disease occurs frequently in inherited syphilis, and is often first discovered when the cornea clears from an attack of interstitial keratitis. In exceptional cases there is no deposition of pigment in the atrophic stage. Where the disease is not arrested early, the atrophic spots merge into one another, and thus a large area of the sclera is exposed. Patches which ap-

parently have undergone complete atrophy may continue to enlarge without the advent of fresh exudation. In a late stage of chorioiditis masses of connective tissue form in the retina, simulating retinitis proliferans; in the latter, however, the exudation lies in front of the retinal vessels. Vitreous opacities may be found at any stage of chorioiditis. They are frequently small and "dust-like," particularly in the syphilitic form of the disease, but may be large and thread-like. They may be fixed or movable. The lens, at this stage, begins to show opacity, particularly at its posterior pole, from which striæ radiate. Adhesions form between the iris and the capsule of the lens. The eye becomes soft at a late stage and detachment of the retina often occurs. The vitreous may shrink, drawing the lens backward with it and thus greatly increasing the depth of the anterior chamber. The

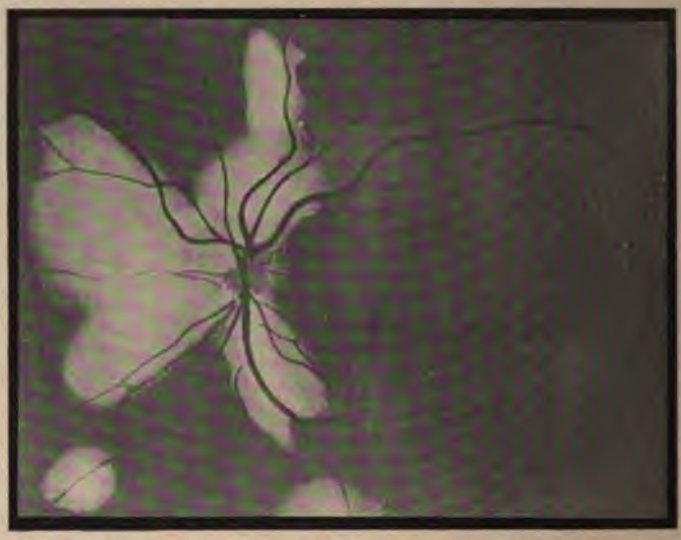


Fig. 286.—Early stage of chorioidoretinitis. (LIEBREICH.)

formation of bone is not unusual in such eyes. The term *atrophy of eyeball* is applied to such shrunken globes. Keratitis punctata may be present at any stage of chorioiditis, indicating that the disease has reached the anterior portion of the uveal tract.

CLINICAL FORMS OF EXUDATIVE CHORIOIDITIS.—The classification of inflammations of the chorioid is an unsatisfactory matter. Ophthalmic writers have made many divisions, based either upon the location of the morbid process or on its supposed etiology. The following clinical forms are recognized:—

1. Disseminated chorioiditis.
2. Central chorioiditis.
3. Myopic chorioiditis.
4. Syphilitic chorioidoretinitis.
5. Anterior chorioiditis.
6. Chorioiditis with descemetitis.
7. Gonorrheal iridochorioiditis.

1. Disseminated Chorioiditis (Fig. 2, Plate XIV) may involve one or both eyes; in the latter instance one eye is usually much more involved than the other. Scattered over the fundus will be found alterations whose appearance will depend upon the stage of the disease. In recent cases the fundus will show numerous rounded or stellate elevated patches of a yellowish-white or grayish color, over which the retinal vessels course. In old cases the spots will be whitish, from exposure of the sclera, with pigmented borders. The spots may coalesce to form large patches, which present a punched-out appearance. The disease is chronic, new patches being formed from time to time. In an eye which is studded with such atrophic spots central vision may remain good, if only the macula is spared. The optic nerve is affected

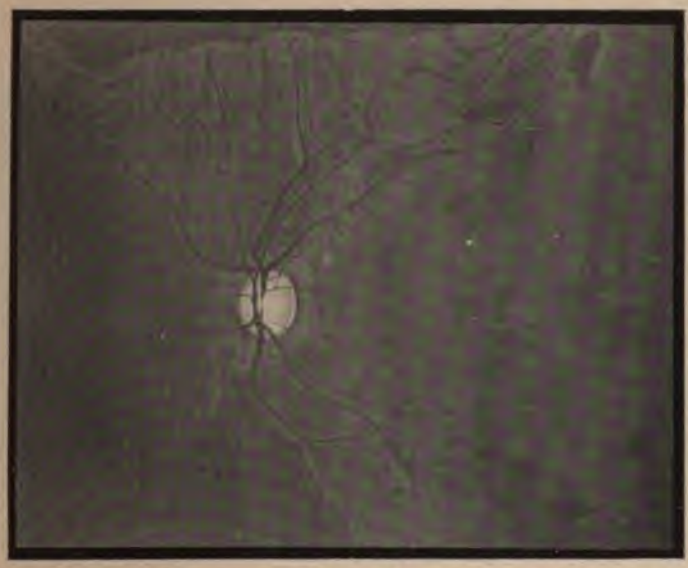


Fig. 287.—Late stage of chorioidoretinitis. (LIEBREICH.)

in disseminated chorioiditis. In the early stage both the retina and nerve-head are hyperemic, while later they become atrophic. The papilla loses its clear outline, becomes of a dirty-grayish color, and the retinal vessels are fewer in number and much contracted. Vitreous opacities, dust-like or string-like, are frequently present.

Förster, in 1862, described a form of disseminated chorioiditis under the name *areolar chorioiditis*, in which foci appear in the macular region and subsequently others come at increasing distances therefrom. At first each focus is black; gradually the pigment is removed from the centre toward the periphery, and finally the spots become almost white.

2. Central Chorioiditis.—Inflammation of the chorioid in the region of the macula may be found at any period of life. When bilateral and occurring in infants it has an injurious effect upon the mental development

of the child. The eyes are amblyopic and nystagmatic; and the facial expression is suggestive of idiocy. Older subjects will complain of loss of vision and the presence of a central scotoma. Ophthalmoscopic examination shows the presence of an exudation in the macular area, if the case is seen early; if examined at a later period, a large, white, atrophic area, bordered with pigment, is found. The disease, both in the infantile and the senile forms, is sometimes found in several members of a family. In elderly persons the ophthalmoscopic signs may consist of a round or oval area at the macula, presenting yellowish, erosion-like dots and pigmentary changes, minute hemorrhages, and crystals of cholesterin; or the macula may look as though salt and pepper had been dusted upon it. Hutchinson and Waren Tay described a condition of the fundus, known as "Tay's

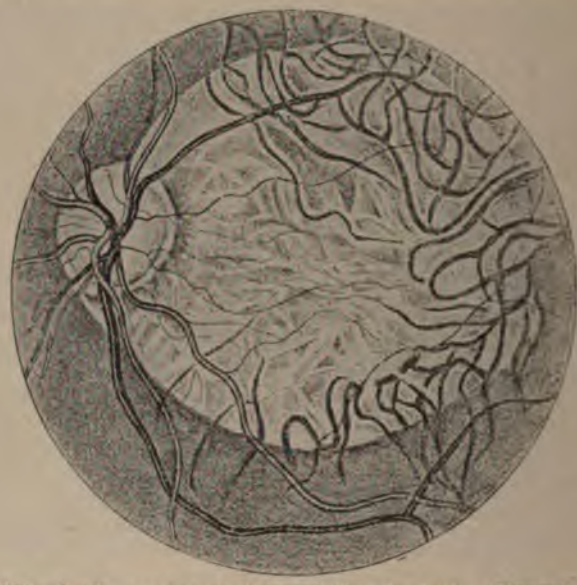
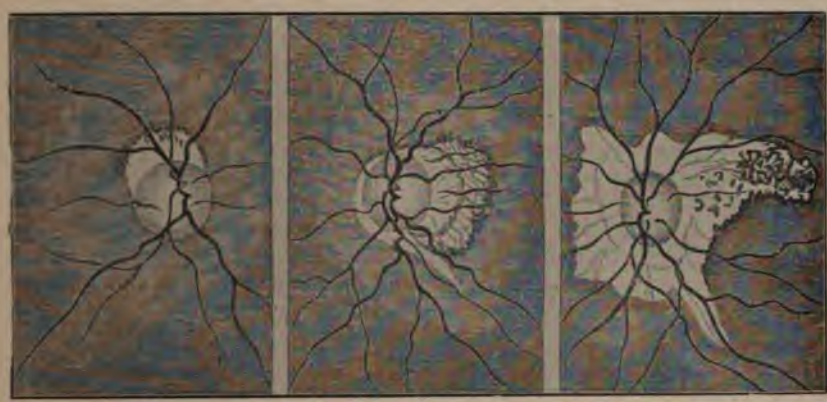


Fig. 288.—Senile areolar atrophy of the chorioid. (NETTLESHIP.)

chorioiditis," or "central senile guttate chorioiditis," in which the macular area is filled with closely set, pale-yellowish dots. This condition, which was supposed to be found only in middle-aged and elderly persons, has been observed at all ages, and is similar to punctate conditions found in the retina. It is to be distinguished from colloid disease of the macular region. Another form of central chorioiditis has been named senile areolar atrophy of the chorioid, in which, owing to atrophy of the pigment layer, the stroma of the chorioid and the curves and anastomoses of its vessels can be plainly seen. Such a case may present the vessels as atrophic white lines (sclerosis of the chorioidal vessels). The failing vision of elderly persons in whom the dioptric media are clear is often due to central chorioiditis; and this condition may be a cause of great disappointment after a technically successful cataract extraction.

3. Myopic Chorioiditis.—While, for the sake of convenience, the term myopic chorioiditis (Fig. 2, Plate XVII) is retained, it should be understood that in the majority of cases of myopia with chorioidal changes the process is a mechanical one, producing atrophy, inflammatory symptoms being of rare occurrence. In axial myopia the antero-posterior diameter of the globe is elongated by thinning of the posterior segment of the sclera. Although a limited amount of small-cell infiltration is present, the chorioidal condition is not classed as inflammatory. The change in the sclera can be seen ophthalmoscopically and demonstrated microscopically. The fundus changes of myopia can be best considered under two heads: (1) posterior staphyloma and (2) myopic degeneration.

(a) Posterior Staphyloma (Posterior Sclerochorioiditis).—This term is applied to a condition often accompanying myopia, the chorioid adjacent to the nerve-head becoming atrophic. The stretching of the sclera is indi-



1. 2. 3.

Fig. 289.—Types of posterior staphyloma. (GALĘZOWSKI.)

1, Superior staphyloma. *2*, External staphyloma. *3*, Annular staphyloma.

cated by the appearance of a crescent which is usually found on the temporal side of the disc, but sometimes is seen inferiorly, superiorly, or nasally. The crescent is made by the sclera, which has been stretched, and, since the chorioid cannot follow, the white sclera lies exposed. This explanation is questioned by Schnabel (see "Myopia" in Chapter XXI). The exposed part may assume a triangular shape, and then is called *conus* (Jaeger). This term, however, is confusing, and should not be applied to the condition found in myopia, but should be reserved for that congenital anomaly in which the nerve-head does not fit accurately into the chorioidal aperture. If the atrophy of the chorioid spreads around the nerve-head, the condition is called *annular staphyloma.* Since the boundary between the nerve-head and the staphyloma is often ill defined, the beginner in ophthalmoscopy may mistake a staphyloma for an unusually large papilla. The differentiation between the two is made by the greater redness of the nerve-head. The

line separating the chorioid from the staphyloma is better defined, is irregular, and is often bordered with pigment. The vessels emerging from the nerve-head are often narrow, straight in their course, and look as if they were stretched. The fundus changes may be limited to the vicinity of the nerve-head, but generally are more extensive. Rarely posterior staphylomata are found in hypermetropic and emmetropic eyes. Ophthalmoscopic examination of a highly myopic eye with posterior staphyloma will show the "Weiss reflex." This appears on the nasal side of the papilla, at a distance of several disc diameters, as a curved line which presents a narrow light streak bordered by two dark lines (Fig. 2, Plate XX). Its presence indicates the existence of a posterior scleral ectasia.

(b) Myopic Degeneration.—This term has reference particularly to those fundus changes which occur in and around the macula lutea. In cases of progressive myopia there is not only posterior staphyloma, but marked changes are present in the entire fundus; and in malignant cases the changes involve even the ciliary body, vitreous, and lens. Loss of pigment in the epithelial layer permits the chorioidal vessels to become visible. Accumulations of pigment are often found as black spots near the macula. Hemorrhages may appear in the same locality, and after resorption large or small white areas bordered with pigment are to be seen. The fundus may present a tessellated appearance. Central visual acuity is much reduced. Opacities appear in the vitreous and lens (posterior polar cataract), and detachment of the retina sometimes occurs.

4. Syphilitic Chorioidoretinitis (Fig. 1, Plate XIV) will be considered in Chapter XV, under the term "Syphilitic Retinitis."

5. Anterior Chorioiditis.—This term is applied to that condition in which the anterior portion of the chorioid is the site of exudation. The changes may be readily overlooked unless this part of the eye is carefully examined with the ophthalmoscope. The most frequent causes of anterior chorioiditis are myopia and hereditary syphilis. Doubtless the disease frequently accompanies parenchymatous keratitis. In cases permitting an ophthalmoscopic examination the periphery of the fundus shows numerous rounded black spots. In elderly persons ordinary pigment changes are often found in the anterior portions of the chorioid.

6. Chorioiditis with Descemetitis.—A. Hill Griffith has called attention to a form of chorioiditis occurring in young subjects, chiefly females, and characterized by the appearance of dust-like opacities of Descemet's membrane. The disease begins suddenly with failure of vision of one eye and without external signs of inflammation. The iris is normal. Ophthalmoscopically a patch of recent exudation, of a whitish or bluish-gray color, is found in the chorioid and retina adjacent to the optic disc. Fine opacities are sometimes present in the vitreous, and shreds of fibrin may extend into it from the chorioidoretinal patch. The dots on Descemet's membrane are supposed to be derived from the chorioid. The disease occurs chiefly in tubercular subjects. Ozena is a frequent accompaniment. In

PLATE XIV.

Diseases of the Choroid.

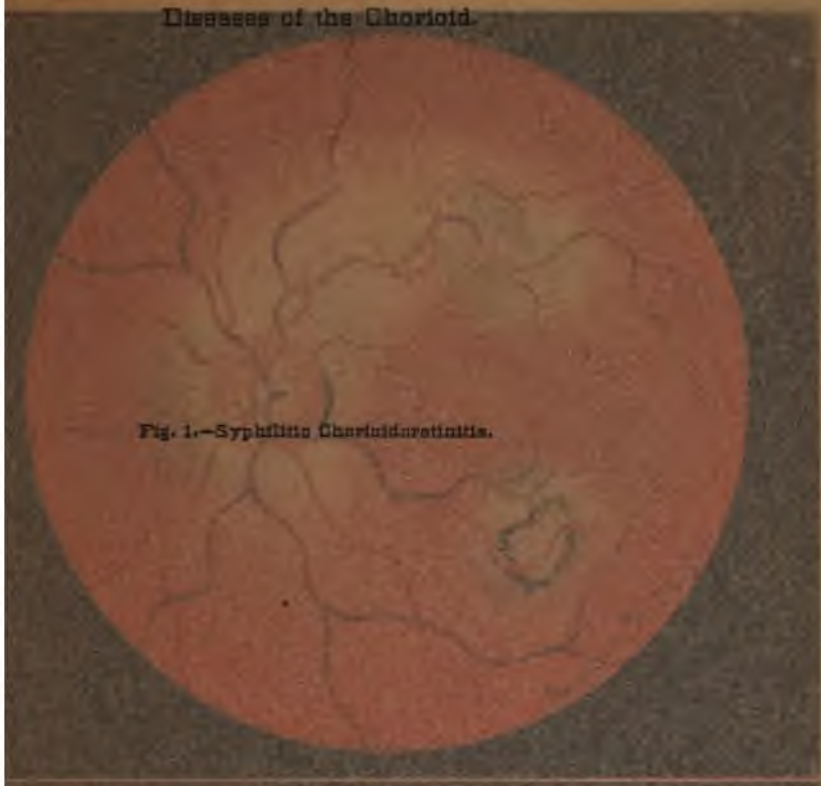


Fig. 1.—Syphilitic Choroidoretinitis.

Fig. 2.—Disseminated Choroiditis.

th's cases syphilis was not present. The prognosis is favorable, the se disappearing under antitubercular treatment.

r. *Gonorrheal Iridochorioiditis.*—Bull has described cases of inflamma- of the uveal tract which followed or were associated with gonorrheal itis. The ocular symptoms were severe, and included pain, photo- a, lacrimation, and great reduction in visual acuity. Ciliary injection, ıtion into the anterior and the vitreous chambers, and increase of intra- r tension were noted. Vision, which may be reduced to perception of improves rapidly after the administration of antirheumatic remedies.

Causes of Exudative Chorioiditis.—While many cases of exudative oiditis are doubtless due to syphilis, and others are attributed to rheu- sm, gout, tuberculosis, anemia, and other diathetic diseases, there ins a large percentage of cases in which no general disease can be ted. Trauma, in the form of penetrating wounds, operations, or s without visible lesion, accounts for some cases. Occurring in young cts who otherwise are in good health, chorioiditis must be attributed ıently to excessive use of the eyes in near vision, either with or without roper correction of errors of refraction. Disseminated chorioiditis is times an hereditary affection, and at times it is found accompanying enital cataract.

Woods, of Baltimore, states that among the causes of chorioidocyclitis lisorders of menstruation, intestinal affections, naso-pharyngeal disease, acute infections.

Pathology.—At the beginning of the process hyperemia and edema resent, and are soon succeeded by a serous or fibrinocellular exudation. may be found between the chorioid and retina, on the surface of the a, or in the vitreous humor. Exudation present in the vitreous humor ca use punctate opacities or membranous bands. Edema of the retina hemorrhages are frequent accompaniments of chorioiditis. The acute is succeeded by a period in which the chief features are the formation w connective-tissue fibres, the disappearance of the smaller chorioidal ls, atrophy of the chorioid and retina, adhesion between these mem- s, and the proliferation of pigment epithelium. The affected structures hen represented by a transparent cicatrix, which is bordered with pig-

Diagnosis.—The diagnosis of chorioiditis must be made ophthalmo- cally. Often, however, it will be difficult to determine whether the logic process is chiefly in the chorioid or in the retina. Pigment gations resembling bone-corpuscles are always in the retina (Nettle- . Exudates attributed to the retina often cover the retinal vessels, chorioidal exudates are situated under the retinal vessels. Accord- to Meyer, the retinal exudates are more opaque than are those of the ioid, and their borders show fine radiating lines, corresponding to the ction of the nerve-fibres. In retinitis the retinal vessels are tortuous. chorioiditis diffuse opacities in the vitreous humor may so veil the

retinal vessels as to simulate retinitis. Differentiation between a recent chorioidal exudation and old atrophic spots is important. In *recent chorioiditis* the ophthalmoscopist can see an exudation of yellowish-white color, with ill-defined outlines. No chorioidal vessels are visible. The retinal vessels pass over the exudation and are seen to bend. The points of exudation are free from pigment. Cases of *old chorioiditis* show pure white atrophic areas of irregular outline, often with a border of pigment; frequently islets of pigment exist within the atrophic area, and in rare cases the migration of pigment continues until the spot becomes black. In such an event the movement of pigment is from the chorioid into the retina, as can be proven in microscopic sections. The remains of chorioidal vessels are sometimes visible within the white atrophic areas, and not infrequently the vessels show white, thickened walls, or complete obliteration, the vessels then appearing as white cords. Differentiation between coloboma and old chorioiditis may be difficult, especially between the rounded coloboma occurring at the macula and elsewhere and the isolated patch of chorioiditis. The history of the case and the fact that in coloboma other anomalies are generally present will aid in the diagnosis. In some instances only the pigment layer of the chorioid is involved in atrophy. The pigment gradually disappears, thus permitting the chorioidal vessels to appear and form a tessellated fundus. This type of atrophy is common in myopia, in glaucoma, and in retinitis pigmentosa, and is physiologic in old age.

Prognosis.—Exudative chorioiditis is a serious disease, frequently ending practically in blindness produced by the recurrence of acute inflammatory attacks. The disease will affect vision in proportion to the proximity of pathologic changes to the macula. While complete recovery is never to be expected, the prognosis is much more favorable in the chorioiditis due to eyestrain occurring in young subjects than in chorioiditis due to syphilis or to unknown causes in old persons.

Treatment.—A patient with exudative chorioiditis should be given atropin and should wear dark, smoked glasses to protect the eyes from bright light. Under these he should wear the proper glasses to correct his error of refraction. Use of the eyes for near work is to be prohibited. Except in severe cases of iridochorioiditis, confinement in a dark-room or in bed is unnecessary. In case chorioiditis occurs in young persons with eyestrain, who present no signs of syphilis, the internal treatment is limited to the occasional use of an aperient and the daily administration of ferruginous tonics. This plan of medication, combined with rest of the eyes for a period of several weeks, and the prescription of proper glasses, will often result favorably. This is particularly true of cases in which the exudation is limited in area. In cases of disseminated chorioiditis it is advisable to place the patient quickly under the influence of mercury, and preferably by inunction. Iron also is often required, and complete mental and physical rest is to be advised. In all recent cases atropin and dark glasses are to be used. Iodid of potassium is a valuable remedy, though

probably inferior to mercury. These internal remedies are to be continued in milder doses long after the exudation has disappeared. Patients in whom signs of syphilis are present will probably receive much more benefit from this plan of treatment than will those in whom the diagnosis of syphilis cannot be established. In non-syphilitic chorioiditis some surgeons report good results from the use of salicylates. Cases of chorioiditis are recorded in which no benefit followed mercurial inunctions, and great improvement was apparent after subconjunctival injections of bichlorid of mercury or the hypodermic injection of pilocarpin. In old cases of chorioiditis strychnin and galvanism may be tried.

Purulent Chorioiditis is divisible into endogenous and ectogenous forms. The endogenous variety is of rare occurrence and is produced by the lodgment of septic emboli in the ocular vessels, by the transfer of meningeal inflammation, or by the transfer of inflammation in orbital phlegmon and in thrombosis of the orbital veins. The ectogenous form is of frequent occurrence. It follows upon ophthalmic injuries and operations, and is found after perforating corneal ulcers and in cases of iris-prolapse. The varieties of purulent chorioiditis will be discussed under the names, respectively, of "Endogenous" and "Ectogenous Panophthalmitis."

Endogenous Panophthalmitis (Metastatic Chorioiditis).—Inflammation of the chorioid by metastasis fortunately is of rare occurrence. It has been found during cerebro-spinal meningitis, typhoid fever, scarlatina, puerperal fever, erysipelas, mumps, caries of the cranial bones, ulcerating endocarditis, septicemia following surgical operations and compound fractures, and in the course of pneumonia due to influenza. The existence of purulent chorioiditis as a metastatic affection was established by Virchow in 1856. Both eyes are usually affected, one after the other. Bull, of New York, who has carefully studied six cases occurring in the course of grippe-pneumonia, and has made two autopsies, states that the microörganisms found in such cases are the staphylococcus albus and aureus, the streptococcus pyogenes, and the pneumococcus. Bacterial thrombi have been demonstrated in the vessels of the iris, ciliary body, chorioid, retina, conjunctiva, ocular muscles, and orbital tissue.

Purulent chorioiditis occurring in the course of meningitis is attributed to direct extension of the morbid process along the lymph-spaces of the optic nerve. It occurs chiefly in children, and in the course of epidemic cerebro-spinal meningitis.

Post-partum metastatic chorioiditis usually appears between the sixth and fourteenth days after delivery, and is more frequently unilateral than bilateral. It is a sign of the gravest import. Often there is rapid loss of vision with little pain and few external signs of inflammation. Circumcorneal injection, iritic adhesions, hypopyon, with slight pain, are soon followed by the symptoms of acute panophthalmitis, perforation of the globe, a discharge of pus, and shrinking of the eye. Of sixty-three cases collected by Axenfeld, twenty-two were bilateral.

Symptoms.—The disease is announced by headache, vomiting, rise of temperature, pain in the eye and head, with the ordinary signs of iridocyclitis, and rapid and total loss of vision. Intra-ocular tension is at first increased, but later becomes decreased. The disease runs its course in from three to six weeks and ends in blindness, with shrinking of the globe. Ophthalmoscopic examination very early in the case will show numerous foci of exudation into the chorioid and retina. In a few hours the media will be clouded. The vitreous will be filled with fine opacities, the fundus presenting a dense, yellowish reflex. Iritis, exudation into the pupil, chemosis, and sometimes exophthalmos rapidly follow. Early perforation at the corneoscleral junction is not uncommon.

Diagnosis.—The diagnosis of metastatic chorioiditis is not usually difficult, provided the surgeon obtains the history of the case and makes an ophthalmoscopic examination. The more severe cases, with pain, iridocyclitis, and conjunctival chemosis, are to be spoken of as examples of metastatic panophthalmitis, while those of less severity, in which the lesions are confined to the fundus, are known as cases of metastatic chorioiditis.

In the case of children, when purulent chorioiditis follows meningitis or thrombosis of the orbital veins, the ophthalmoscopic picture may resemble that of neuroepithelioma of the retina. Usually, however, the presence of iritic adhesions, diminished tension, the anterior chamber being deep at the periphery and shallow at its centre, together with shrinking of the globe and the usual absence of vessels upon the intra-ocular mass, will serve to distinguish pseudo- from true neuroepithelioma. The diagnosis is further discussed in the chapter on the "Retina." In case of doubt it will be best to excise the eye.

Prognosis and Treatment.—Since metastatic chorioiditis occurs in the course of a grave systemic disease, often producing death, the prognosis is most unfavorable. If life is saved the affected eye usually remains blind. In some instances the chorioiditis due to meningitis runs a mild course and leaves the patient with a small amount of vision. Metastatic chorioiditis following puerperal septicemia gives a most unfavorable prognosis. Of nine cases observed by Hirschberg, all died; recoveries, however, have been recorded by Kipp and Wood. Kipp's case was unilateral and Wood's was bilateral. In the stage of acute suppuration, in metastatic chorioiditis, Bull performs evisceration for the relief of pain, but advises against enucleation, owing to the possible danger of producing meningitis. The diagnosis, however, must be clear. Less heroic measures for the relief of pain are the application of hot compresses and the administration of narcotics. When the eye becomes shrunken, it should be removed, if tender to the touch.

Ectogenous Panophthalmitis.—This term indicates an acute suppurative inflammation of the eyeball caused by trauma, by infection following the perforation of a corneal ulcer, or by infection following prolapse of the iris. In other words, the infection is from without. Most

cases of panophthalmitis are the result of accidental injuries, such as penetrating or lacerated wounds, with or without the lodgment of a foreign body. Exceptionally the disease occurs after operations made upon the eyeball for cataract, or glaucoma, or after an optical iridectomy. The frequency of such disasters will vary with the practice of different surgeons.

Symptoms.—Ectogenous panophthalmitis is characterized by pain, rapid and total loss of vision, and the objective signs of an intense inflammation. The pain is often excruciating and involves the eye and the head. Constitutional disturbances, such as nausea and vomiting, chills, and rise of temperature, are present. In from twenty-four to forty-eight hours after infection the objective signs are unmistakable. The eyelids are swollen; the tissues supplied by the pericorneal and conjunctival vessels are intensely hyperemic; the chemotic conjunctiva may protrude beyond the eyelids; the

Fig. 290.—Panophthalmitis and orbital cellulitis. (W. T. Shoemaker.)

cornea is cloudy and the aqueous is muddy or purulent. The iris is lustreless, hyperemic, and adherent to the lens-capsule. The eye is tender to the touch, immobile, and shows some exophthalmos. The tension is increased until after the occurrence of perforation, which takes place at the site of the wound (Fig. 6, Plate XI). The globe shrinks and forms a small, deformed stump. If unchecked by operative intervention, the entire process occupies many weeks. If the infection begins in the depths of the eye after the lodgment of a foreign body, the aqueous and cornea may remain clear for some time and the iris may be movable. In such cases ophthalmoscopic examination shows a yellowish deposit deep in the vitreous. With a dilated pupil a yellowish reflex is seen by ordinary inspection.

Pathology.—Microscopic examination shows the chorioid and retina filled with round-cell infiltration, and in many cases these structures are obliterated, their place being occupied by areas of round cells, coagulated

material, and pigment granules. The veins show thrombosis, the arteries contain emboli, and the smaller vessels present hyalin swelling. Various forms of streptococci and staphylococci are present. Macroscopically it will be observed that the enucleated globe scarcely resembles the eyeball, owing to the great thickening of the sclera. The separate structures of the eye have lost their individuality, and the thickened scleral cup contains pus, blood, and *detritus*. The thickening involves the sclera, the optic nerve, and its sheaths. Tenon's space is obliterated by adhesion of the capsule to the sclera, and thus exophthalmos is produced.

Diagnosis.—Ordinarily there can be no doubt about the nature of the disease. The history of the case will generally leave no question as to diagnosis. Chemosis of the conjunctiva and swelling of the eyelids due to purulent or to diphtheritic conjunctivitis will present a profuse conjunctival discharge. In panophthalmitis such a discharge is absent. Orbital phlegmon, tenonitis, thrombosis of the cavernous sinus, and panophthalmitis all present swelling of the lids, conjunctival chemosis, exophthalmos, and immobility of the globe. Panophthalmitis, however, shows suppuration

Fig. 291.—Section of a panophthalmitic eye. (WÜRDEMANN.)

within the globe, while in the other affections, which have been mentioned, the anterior part of the eyeball is of normal appearance.

Prognosis in panophthalmitis is distinctly bad as far as vision is concerned, but is favorable as relates to life and to the integrity of the other eye. Recent experiments in intra-ocular disinfection which were made by Ostwalt upon rabbits, and some clinical observations by Haab, who successfully employed iodoform intra-ocularly to check suppuration, seem to show that the prognosis of panophthalmitis will be more favorable in the future than it has been in the past.

Treatment.—A patient with panophthalmitis should be sent to bed. Anodynes should be given to relieve pain. Atropin should be instilled thrice daily and laxatives administered. If a foreign body is present, it should be removed. As regards the treatment of the eye, one of the following procedures may be employed: (1) intra-ocular disinfection by iodoform may be tried, (2) the suppurating eye may be regarded as an abscess and treated by incision, (3) incision may be combined with excision of the cornea and evisceration of the contents of the globe, or (4) an enucleation may be made.

1. The introduction of small pieces (one and one-half to two milli-

metres thick) of iodoform into the anterior chamber has been successful in curing intra-ocular infection which otherwise would have resulted in panophthalmitis and loss of the eye. If a foreign body is present it should first be removed. Haab's success has been confirmed by the experience of others (Goldzieher and Roemer). The iodoform should be specially prepared by sterilization and is introduced by an instrument passed through a wound made in the cornea. The observations concerning this method of treatment are too few to permit of final judgment as to its value.

2. Incision of the panophthalmitic globe relieves pain and shortens the duration of the disease. The globe can be opened with a knife, or, as de Lapersonne prefers, with the galvanocautery. The eye is treated like an abscess.

3. Incision, with excision of the cornea and evisceration, is a favorite method with those surgeons who fear meningitis after enucleation. The cornea is removed and the interior of the eye is cleansed by forceps wrapped with gauze, thus removing the remains of the retina and uveal tract. Gauze is placed in the scleral cup and the eye is dressed daily. This method results in the formation of a small stump, over which an artificial eye can be worn.

4. Enucleation in panophthalmitis is practiced by many surgeons, but is violently opposed by others on the ground that it may cause meningeal infection. While the author does not hesitate to enucleate panophthalmitic eyes, he feels the last word has not been said regarding this operation. Meningitis, however, has been known to occur after evisceration, and also has followed panophthalmitis which was not treated by operation.

INJURIES OF THE CHORIOID.

Wounds of the Chorioid occur in connection with wounds of the sclera, and should receive careful treatment. The chorioid should not be sutured, but the sclera and conjunctiva can be sewed, careful attention being given to cleanliness. Although the primary result may be encouraging, secondary retinal detachment often occurs.

Foreign Bodies lodging in the chorioid should receive the treatment mentioned in the chapter on the "Sclera" (page 371).

Chorioidal Hemorrhage may occur without rupture of the retina, as a result of trauma or idiopathically in the early stage of an exudative chorioiditis. The hemorrhage may be into the structure of the membrane or between it and the sclera. The blood forms a bright-red or brown, oval or circular spot, over which retinal vessels run. The blood gradually becomes absorbed, leaving a white, atrophic spot, with some pigmentation. Acute glaucoma may be produced by chorioidal hemorrhage. Differentiation between hemorrhage into the superficial layers of the chorioid and that into the deeper layers of the retina is impossible. Hemorrhage into the nerve-fibre layer of the retina presents a flame-shape in marked contrast to the

rounded or oval patch of chorioidal hemorrhage. In a case of hemorrhage under the author's care the condition was caused by a blow, and appeared as a circular black spot, one-eighth the size of the optic disc, situated immediately below the macula. Six years after the injury vision was $^{20}/_{30}$. The treatment includes rest, atropin, and smoked glasses, with diaphoretics and mercurials.

Detachment of the Chorioid, aside from being caused by malignant tumors, is probably of much more frequent occurrence than would be supposed from the limited literature. It is caused by trauma, chronic inflammatory processes with shrinking of the vitreous, chorioidoretinitis with adhesions between the two structures, and serous effusions between the chorioid and sclera. It may follow the sudden reduction of intra-ocular tension, and is one of the unfortunate accidents of glaucoma and cataract

Fig. 292.—Ruptures of the chorioid. (KNAPP.)

The figure at the left shows a vertical tear at the left of the optic disc and an exudation at the right. The figure at the right presents two ruptures of the chorioid and a hemorrhage in the region of the macula. In both illustrations retinal vessels are seen crossing unbroken over the ruptures.

operations. The detached portion forms a rounded, brown, smooth mass, resembling a vesicle, but without the folding and flapping of the mass which are features of retinal detachment. Secondary detachment of the retina may occur in these cases. Eyes in which the chorioid is detached often end in atrophy of the globe. The treatment of the condition is rest by atropin and the use of the bandage.

Rupture of the Chorioid follows severe injuries, particularly concussions and blows with blunt instruments, but in many cases it arises from blows that are apparently insignificant; and is often obscured by hemorrhage into the vitreous. After this clears the tear in the chorioid can be seen as a long, narrow, often sickle-shaped separation. According to Knapp, it is rarely combined with laceration or detachment of the retina. It is usually found at the posterior pole, since here the chorioid is bound to the sclera by the entering ciliary vessels, and generally is found on the

temporal side. Its direction in most cases is vertical, although horizontal tears have been observed. The rupture at first is yellowish, but later becomes white. Its margins are bordered by pigment, and often the retinal vessels can be seen running across the break. The damage may end with the rupture, or the chorioid may undergo a chronic atrophic process. When located near the macula, rupture of the chorioid damages vision. Small ruptures are said sometimes to unite spontaneously; large ones always remain. The treatment is atropin, aperients, the application of leeches to the temple, and rest. Often other lesions of the eye occurring at the same time are regarded as of greater moment, and many of these cases are diagnosticated as traumatic iridocyclitis, the rupture in the chorioid being either entirely overlooked or discovered late in the history of the case. There is often dilation of the pupil, paralysis of accommodation, hemor-

Fig. 293.—Colloid change in the macular region. (De Schweinitz.)

rhage into the vitreous, scotoma, and great reduction in visual acuity. Not infrequently vision improves for some weeks after the injury and then becomes permanently bad, owing to contraction of the scar-tissue formed in the chorioidal gap: a fact which bears directly on prognosis. Detachment of the retina sometimes occurs late in these cases.

Colloid Disease of the Macular Region.—A rare condition of the macular region, which is symmetric and which may be mistaken for central chorioiditis, is the occurrence of colloid formations resembling those found in the nerve-head. In a case observed by the author both maculæ were occupied by rounded, yellowish bodies lying beneath the retinal vessels, and presenting a mulberry-like appearance. The patches were oval and equal in length to two disc diameters. The patient was a man, aged 41 years, and vision was normal. Three similar cases have been seen by de Schweinitz. Nettleship has described a similar case as central, guttate

chorioiditis with normal vision. His patient showed "a number of small, perfectly circular, pale, grayish-yellow spots thickly congregated at the yellow-spot region, and more thinly scattered all around that part, reaching on the nasal side as far as the disc." According to Dimmer, the change is a simple colloid degeneration. The excrescences are located in the lamina vitrea of the chorioid. The condition does not call for treatment. Chorioiditis can be excluded by the fact that vision is normal.

Atrophy of the Eyeball.—Plastic inflammation of the uveal tract (iridochorioiditis) often ends in a condition in which the eyeball becomes soft and lessened in all its diameters. The globe is irregular in shape from wrinkling of the sclera. The retina becomes detached. When the exudation lies chiefly behind the lens, the anterior chamber will become shallowed. If the force of the contracting exudate is exerted more in a backward direction, the chamber will be deepened. The condition is known as atrophy of the globe, and is irremediable. Often the optic nerve also becomes atrophic. An atrophic globe, on section, presents the various tunics much altered. Such an eye may be tender upon pressure, and may cause sympathetic irritation or sympathetic ophthalmitis. If tender, the atrophic globe should be removed.

Phthisis Bulbi.—This term is applied to shrunken globes in which the shrinking follows panophthalmitis with perforation of the sclera or cornea. The process is a rapid one, the diminution in size being brought about by a partial loss of the intra-ocular contents and by contraction of exudates. While in atrophy the altered membranes can be distinguished, in phthisis bulbi the retina and uveal tract are destroyed by suppuration. The stump in phthisis bulbi is usually small and innocuous. Hence an enucleation is not usually necessary. If, however, the stump is tender on pressure, it should be removed. As in atrophy, so in phthisis, the optic nerve becomes atrophic.

Ophthalmomalacia (Essential Phthisis Bulbi).—An obscure and rare condition, which was described by von Graefe, is ophthalmomalacia. In this disease diminution of intra-ocular tension, pain, photophobia, miosis, partial ptosis, and rapid shrinking of the globe occur without hyperemia and without assignable cause. The disease may be intermittent and sometimes has followed injuries. It is probably dependent on a lesion in the sympathetic nerve. Fuchs states that the prognosis is favorable. Treatment must be limited to improvement of the general health.

CHAPTER XIII.

DISEASES OF THE CRYSTALLINE LENS.

The crystalline lens is subject to congenital anomalies, to opacities, and to injuries. Being a non-vascular body, it is not liable to inflammation. It is greatly influenced by the state of the other ocular structures and by the general condition.

CONGENITAL ANOMALIES.

They generally consist in coloboma, microphakia, lenticonus, aphakia, luxation, and cataract.

Fig. 294.—Binocular coloboma of the lens without coloboma of the iris or chorioid. (Author.)

Coloboma of the Lens is a rare condition, about one hundred and fifty cases having been recorded. Generally only one lens is involved. In most instances the defect is in the lower inner part, which presents an irregular, ragged line, but exceptional cases have been recorded by Schiess, Lang, Bronner, and Payne, in which the defect was outward or upward and outward. Doyne described a case of coloboma of the iris and chorioid, with projection of the corresponding margin of the lens. Commonly, in lenticular coloboma, the iris and chorioid also show colobomata. Iridodonesis is often present, and the patient may show abnormities of other parts of the body. Generally the refraction is myopic. Vision is much reduced and accommodation is weakened or absent.

The cause of coloboma is a subject of speculation. A rational view in regard to it is that of Treacher Collins. He attributes it to the absence

of adhesions between the pars ciliaris retinæ and the lens-capsule in one part of its circumference in early fetal life. In consequence of this there is an absence of the suspensory ligament at that point. Hence, as the eyeball becomes enlarged, that part of the capsule to which the ligament is not attached is not held taut, producing a depression in that situation.

The best treatment is the correction of the refraction. Search should be made for astigmatism.

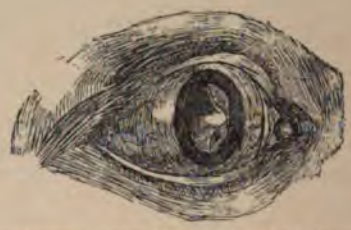


Fig. 295.—Lenticonus anterior.* (Modified from WEBSTER.)

Microphakia.—Cases of abnormally small lens are extremely rare. Reik has reported one with iridodonesis in a rachitic infant. Hartridge has seen a brother and sister with small, transparent, and well-formed lenses. There is a space between the margin of the iris and the periphery of the lens, as is shown ophthalmoscopically with a dilated pupil. Treatment will depend on accompanying conditions. If the lens is opaque it should be needled.

Fig. 296.—Lenticonus posterior in a buphthalmic eye. (Modified from PERGENS.)

Lenticonus.—This is a cone-shaped projection of the anterior, rarely of the posterior, surface of the lens. The refraction is myopic in the projecting area and hypermetropic in the surrounding part. A few cases are on record of lenticonus changing into lentiglobus. The disease is generally unilateral. Anterior lenticonus is supposed to be caused by adhesion of the lens to the cornea in early fetal life, while the posterior form is explained by traction exerted by persistent fetal vessels. The anterior form can be seen by oblique examination and the posterior may be recognized by the peculiar shadow-crescents obtained by the fundus-reflex test; also by the double ophthalmoscopic images of the fundus. Knapp says

that on movement of the mirror blood-vessels and circles and crescents of light and shadow in kaleidoscopic movements will be seen. There is no particular treatment aside from the correction of the error of refraction. In cases of double lenticonus needling of one lens can be tried.

Congenital Aphakia.—Cases of congenital absence of the lens are on record. Dunn has reported one with a central cord in the vitreous humor corresponding with the previous position of the hyaloid artery.

Congenital Luxation of the Lens (Ectopia Lentis) is often an hereditary condition, affecting numerous members of a family. The lens is usually displaced outward, from disturbance of the suspensory ligament. Luxation may exist in company with coloboma of the iris and chorioid, or corectopia may be present. The ophthalmoscope shows double images of the fundus. If the margin of the lens crosses the pupil it will appear as a dark border. The treatment of ectopia lentis consists in prescribing glasses. If the lens occupies the greater part of the pupillary area, the refraction is myopic. Search should always be made for astigmatism. If the lens is opaque, it should be removed by needling or extraction in accord-

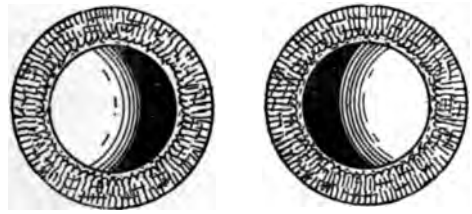


Fig. 297.—Congenital luxation of the lens. (De Beck.)

ance with its condition. A few cases in which the lens remained in the vitreous chamber, attached to the optic nerve, have been reported.

CATARACT.

Cataract is an opacity of the crystalline lens, or of its capsule, or of both. It consists of three main types: capsular, lenticular, or capsulo-lenticular. It may be congenital or acquired. It is either traumatic or idiopathic, and may be partial or complete. It may be primary, or secondary to disease or to an operation; symptomatic of a systemic disturbance; simple or complicated; juvenile or senile; stationary or progressive; hard, soft, or fluid. It may be white, black, brown, or yellow in tint. The location of the opacity in the various forms is shown in Fig. 298.

Senile Cataract (Gray, or Simple, Cataract).—This is often called hard cataract, and appears generally after the forty-eighth year. Sooner or later both eyes become affected. There is a gradually increasing loss of vision. Often a "spider's web" appears before the eyes. If the opacity is central, the patient sees better on a cloudy than on a bright day. If the periphery of the lens is opaque, the reverse is true. In uncomplicated

cases there is not any redness or pain about the eye. As a rule, the patient attributes his defective vision to faulty glasses, objects appearing as through a mist or smoke. In the early stages the lens is swollen, the refraction is increased, and often the patient can see without glasses, giving a condition of so-called "second sight." The increase in refraction may amount to 3 D. Commonly it is from 0.50 to 1 D. In some cases there is a transient hyper-

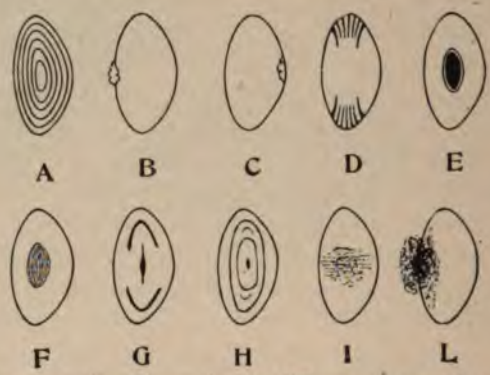


Fig. 298.—Location of opacity in different forms of cataract.

A, Complete capsulo-lenticular cataract. *B*, Anterior polar cataract. *C*, Posterior polar cataract. *D*, Cortical cataract. *E*, Nuclear cataract. *F*, *G*, *H*, Various forms of lamellar cataract. *I*, Axial cataract. *L*, Traumatic rupture of lens-capsule with swelling of lens-substance.

emia of the conjunctiva from eyestrain. Any continued redness of the corneoscleral junction must be looked on with suspicion, and will lead the examiner to seek for more than cataract. Monocular diplopia or polyopia is often present in the formative period. The anterior chamber is ordinarily of normal depth. It may be shallowed, and, if so, it indicates a swollen lens. If it is deepened, it shows that there is a small lens. In uncompli-

Fig. 299.—Anterior capsular cataract of stellate form.
(Von Ammon.)

cated cataract there is not any tremulousness of the iris. The iris should respond to light. If it does not, search should be made for some causative reason, such as iritic adhesions, nervous disease, or morphinism. To the unaided eye the patient's pupil appears dark in the early stage of cataract, or it may present the natural smoky hue of old age; later, except in the rare form of black cataract, the pupil becomes white, gray, or brownish.

The patient's vision gradually diminishes until he can distinguish only light from darkness.

Diagnosis.—Since the lens of the old person naturally has a smoky look, it is always unsafe to make a diagnosis of cataract by ordinary inspection. In suspected cases it is of importance that a correct diagnosis be made early. The catoptric test, the ophthalmoscope, and oblique illumination are all of value in the diagnosis. The catoptric examination is of value for the determination of the presence of the lens and the location of an opacity. It depends on the fact that when a light is held in front of a healthy eye three images of the flame are seen. The first and second, which are erect, are produced by the anterior surfaces of the cornea and lens, respectively; the third, which is inverted, is produced by the concave posterior boundary of the lens. The first and second move in the same direction as the light, while the third moves in an opposite direction. If the lens is opaque, the third image is lost, while the second becomes dimmed; if there is an absence of the deeper erect image, it indicates an opacity of the anterior

Fig. 300.—Appearance of cataract.

A, With reflected, and, *B*, with transmitted light.

capsule. Properly to examine an eye for cataract a weak mydriatic is necessary, preferably a few drops of a 10-per-cent. strength solution of euphthalmin. A stronger mydriatic should not be used, since it may cause an attack of glaucoma. In the examination, light from a concave mirror should be thrown into the dilated pupil, from a point distant about one metre, or closer. If black striæ or spots appear on the red fundus reflex, there is an opacity probably in the cornea, lens, or vitreous. The location of the opacity can be determined by the parallax test, by oblique illumination, or by direct ophthalmoscopy. If the lens is almost wholly opaque, the red reflex from the fundus will be correspondingly reduced. If the lens is wholly ripe, the reflex will be absent.

An incipient cataract may present numerous small, round, black dots instead of black lines on a red field. The condition of the fundus should be determined, if possible. If the cataract is mature there is no shadow between the lens and iris-margin, such as that shown in Fig. 301; and no red reflex when light is thrown directly into the eye. Glaucoma is sometimes called cataract, with fatal result to vision.

In glaucoma there is corneoscleral redness, increased tension, a dilated pupil, and often a beginning opacity of the lens. A practitioner unfamiliar with diseases of the eye may call such a case cataract, advise the patient to wait until the lens is ripe, and thus consign the poor victim to blindness. Where the media are clear, the ophthalmoscope shows glaucomatous excavation of the nerve-head, with arterial pulsation either spontaneously present or produced by slight pressure. Pain is often present in glaucoma.

COURSE.—Cataract is usually progressive, but may remain stationary for many years. This is particularly true of the cortical form. The period between incipiency and full maturity varies from one to three or more years. The opacities may begin centrally or peripherally as clearly defined lines or as dots scattered through the cortex. If a cataract becomes overripe and undergoes liquefaction, the nucleus becoming movable in the capsule, the condition is called a Morgagnian cataract. In these cases the iris is tremulous. Fatty and calcareous changes may occur in old cases.

ETIOLOGY.—In many instances cataract must be regarded as a physiologic process which is incident to age. The frequent occurrence of chorioidal changes in so-called senile cataract is illustrative of pathologic

Fig. 301.—Shadow of iris seen in unripe cataract. (FUCHS.)

states which affect the nutrition of the eyeball. Not infrequently it stands in causative relationship with general disease, such as diabetes, nephritis, etc. Heredity, exposure to great heat, ergotism, arterial sclerosis, uric-acid diathesis, eyestrain, and traumatism are etiologic factors. Often the etiology of cataract will be in doubt. In some cases it must be attributed to the influence of several factors.

PATHOLOGY.—As a general rule, the cataractous lens contains relatively less water and more solids than the non-cataractous. W. J. Collins believes that this dehydration, associated with opacification, is in no sense dependent upon changes which are the result of age, and has no relationship with the ordinary senile ones. The cataractous lens is lighter in weight and is smaller in size than the clear lens. Berzelius considered the chemic change in cataract to be a coagulation of albuminoids and an increase of salts, cholesterin, lecithin, fat, and extractives. In cataract the growth of lens-fibres stops; their separation occurs, producing spaces which become filled with an albuminous liquid. This fluid coagulates and forms granular masses, which are known as the spheres of Morgagni. Some of the lens-fibres become swollen and remain, while others are changed

into vesicle-like cells. The lenticular epithelium thickens, and the cortical lens-fibres disintegrate, while the central fibres remain unchanged.

PROGNOSIS.—The condition of the other ocular structures, particularly the retina and chorioid, as well as the general health, have their influences on the outcome of cataractous lenses. If the rest of the visual apparatus is healthy, the patient, even with a mature cataract, will be able to recognize the location of a distant light in every part of the visual field. If he suffers from disease of the external ocular structures, such as inflammation of the lids, conjunctiva, or lacrimal apparatus, this should be remedied before any operation is performed. Where iritic adhesions are present, an iridectomy should be made. The general condition of the patient is of importance and should be brought as near to normal as possible before operation is undertaken. Age *per se* is not a barrier to successful operation.

TREATMENT.—There are two indications: local therapy and general care. The constitution of the patient should be carefully studied, and errors in diet, method of living, etc., should be corrected. Often these subjects will receive benefit from a course of treatment directed to the intestinal tract. Their tendency to indoor life should be checked. In the early stages of cataract much benefit is derived from a proper correction of refraction. If the lens ripens slowly, in suitable cases one of the operations for artificial ripening can be performed. The use of weak strengths of atropin often temporarily improves vision. There is no medicine which possesses the power of removing cataract. In suitable cases the cataract should be removed by an operation.

Congenital Cataract.—Eyes affected with this condition may present a white pupil, in which case the condition is observed early. It may exist in the form of limited opacities presenting many variations, in which case the child may reach the school-age before visual acuity is markedly diminished. The former type may be classed as complete congenital cataract, and the latter is spoken of as incomplete.

COMPLETE CONGENITAL CATARACT is to be treated by needling, as soon as observed, even as early as the fourth or fifth month. The pupil should be enlarged by a weak mydriatic, a general anesthetic is to be administered, and the anterior capsule is to be opened with a cataract-needle. Repeated operative procedures are safer than a single extensive one. At the first trial only a small opening in the capsule is to be made. After complete subsidence of all reaction the operation can be repeated. After operation the use of atropin is necessary.

INCOMPLETE CONGENITAL CATARACT presents many varieties. The following forms, which are to be attributed to disease of the fetus or to its faulty development, are observed:—

(a) Axial cataract, the condition in which the opacity extends either partly or entirely through the lens in an antero-posterior direction. Other forms of opacity may coexist with it.

(b) Anterior polar cataract, dependent upon adhesions of the pupillary membrane and capsule, or to inflammation.

(c) Posterior polar cataract, existing as a small, white, pyramidal mass situated at a posterior pole of the lens, and due to a persistent hyaloid artery or to some preceding retinochorioidal disease.

(d) Zonular (lamellar) cataract, which is the most common variety, presenting many peculiarities in the form and location of the opacities, the tendency to its development being often hereditary and occurring in scrofulous, rachitic, or feeble subjects, the opacity existing in the layers lying between the nucleus and the cortex.

(e) Punctate and stellar cataract, which have been occasionally observed.

(f) Congenital central or nuclear cataract, which may exist alone or in combination with other forms.

(g) Coralliform cataract, which is a rare variety.

TREATMENT.—If proper glasses give the patient sufficient vision to enable him to obtain an education and follow a vocation, operation is not

Fig. 302.—Coralliform cataract. (AUTHOR.)

to be considered, as often congenital forms of cataract may remain stationary for many years. The choice of operation lies among a simple iridectomy, lenticular extraction, and discission. The iridectomy should be made opposite the clearest part of the lens. If this procedure fails to improve vision, a discission or extraction must be done. In case only one eye is affected with incomplete cataract it is best not to operate, since the removal of the cataractous lens produces such inequality in the refraction of the two eyes that binocular vision is rendered impossible. There are cases in which the operation may be done for cosmetic reasons.

Soft Cataract.—Independent of those opacities, which are congenital and remain unnoticed for long periods of time, there is a form of cataract which appears between the ages of fifteen and thirty years. In such subjects the lens has not formed a hard nucleus. In senile cataract the nucleus practically remains unchanged. In youth it is prone to degenerative changes. It splits, liquefies, and possibly becomes absorbed, ending in calcification of lens *débris*, with capsular thickening. Often while a lens is undergoing degeneration, fat, cholesterin, and lime, with a small quantity of water,

will be found in it. If much lime and considerable fluid be present, the lenticular opacity becomes milky in character, and is known as milky, or *lactate, cataract.* If the entire lens is calcified and shrunken, the condition is named *calcareous cataract.* If intracapsular ossification be present, it is known as *osseous cataract.* If only a thickened capsule remains, it is given the name of *membranous cataract.* Ordinarily, the juvenile form of cataract appears as a large mass of a mother-of-pearl color. The patient may be apparently in perfect health or be the subject of diabetes. Some cases of juvenile cataract are probably due to some such condition as convulsions or a forgotten trauma.

TREATMENT.—This varies according to the condition of the lens. If the lens be of normal dimensions, appearing as a whitish body which completely fills the lenticular space, it should be needled several times at proper intervals. Usually absorption of the lens occurs with a good result. Here, as in other such operations in the young, the use of a cycloplegic and mydriatic is necessary both before and after the procedure.

If the cataract is calcareous or membranous, abstraction of the mass, or an excision of the central portion of it, is the proper procedure, as ordi-

Fig. 303.—Anterior polar cataract. (VON AMMON.)

nary discission will often fail. As it is well to have the patient completely under control, it is advisable to employ a general anesthetic. As a rule, if the patient has one good eye, operation is not advisable except for cosmetic purposes.

Traumatic Cataract.—As its name indicates, traumatic cataract usually arises from the penetration of some foreign body, with or without its retention. It not rarely occurs from a blow which, at the time, does not produce any visible change in any part of the eyeball. Subsequently the lens becomes opaque. The injury may have been given to the anterior or posterior capsule, with or without rupture of the suspensory ligament. The entire lens may have been dislocated and may become opaque. If a foreign body carries infection with it, the case is much complicated, and may pass into a condition of panophthalmitis. A clean foreign body may lodge in the lens without exciting any trouble, being accidentally found years after should the patient be operated on for supposed senile cataract. On the contrary, a portion of the lens which was opaque immediately after the trauma may become transparent.

There is a large number of cases, however, in which such an injury is followed by a non-septic type of inflammation. This is particularly true

of injuries produced by thorns, and other sharp instruments, such as needles or knife-blades, in which the missile is withdrawn at the time of the accident. In a few hours the eye becomes red and injected. There is rapid swelling of lens-substance. At times there is a corresponding increase in intra-ocular tension, which, unless relieved, destroys the eye by secondary glaucoma. Usually there is great pain with corneoscleral redness and chemosis.

TREATMENT.—In the acute type of traumatic cases it is necessary to employ a 1-per-cent. strength solution of atropin, sufficiently often to secure maximum dilation of the pupil. A weaker solution should be used for children. This reduces inflammation and prevents iritic adhesions. The repeated applications of hot wet cloths are useful for the relief of pain. Under no circumstances should any kind of poultice be applied to the eye. It is often advisable to remove all dressings and to protect the eye from light by dark glasses. If intra-ocular tension increases, an immediate operation for its relief will be necessary. In such cases it has long

Fig. 304.—Calcareous cataract. (VON AMMON.)

been the practice of the profession to make a linear extraction, incising the cornea with a lance-shaped knife and removing the lens *débris* through the opening. For reasons which will be given at length in the latter part of this chapter, the extraction of acute traumatic cataract should be done through a large opening made with a von Graefe knife. This procedure enables the operator to remove all of the lens at one sitting. It does not bruise the tissues and gives better results than the linear operation. In cases in which there is not much increase of tension the inflammatory symptoms subside gradually under appropriate treatment. If the patient is young and all signs of inflammation have disappeared, the cataract can be needled. If the case be that of an adult, the lens can be extracted.

Post-operative Cataract.—This is another type of traumatic cataract. In this there is an opacity which is either left after a cataract operation or appears at a later date. In ordinary extraction the anterior capsule is cut to permit egress of the lens. The cut edges retract, and, in favorable cases, leave an unobstructed area. Ordinarily the posterior capsule is not inter-

fered with. At times lens *débris* and proliferating cells of the anterior capsule may remain and form an obstruction to vision. This complication requires a secondary operation in order to insure a betterment of vision. So frequent is the occurrence of this kind of obstruction that the delivery of the lens in its capsule, or a special treatment of the posterior capsule at the time of extraction or a few weeks later, has been advocated. In those cases of extraction in which vitreous humor is lost the posterior capsule is ruptured. In ordinary senile cataract the posterior capsule is so thin that at first it does not form any impediment to vision, but later, usually after one or two years, the opaque capsule materially reduces vision. It is this that has caused the belief that cataract returns after operation.

COURSE AND DIAGNOSIS.—The changes occurring in a capsule after operation are these: The anterior capsule is drawn away from the pupillary

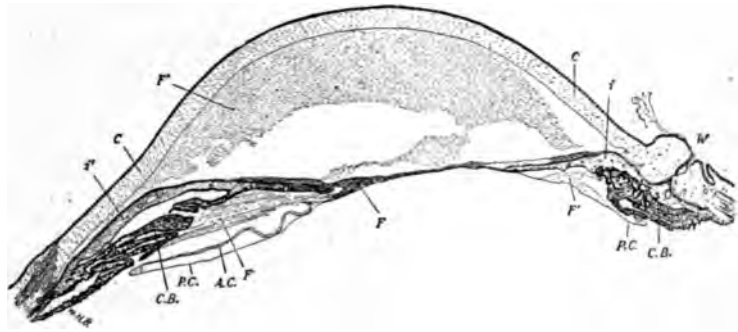


Fig. 305.—Anterior segment of a rabbit's eye twenty-four hours after extraction of the lens. (BATES.)

F, Dense fibrin in the pupillary area. *F'*, Fibrin behind the iris, which is of a lighter structure. *F''*, Fibrin in the anterior chamber. *i*, Iris near the wound. *i'*, Iris opposite the wound. *C.B.*, Ciliary body. *C*, Cornea. *W*, Wound of the cornea; the iris is caught between its inner lips. *A.C.*, Anterior capsule. *P.C.*, Posterior capsule.

area and becomes adherent to the posterior membrane; the capsular epithelium and lens fragments are thus shut off from the action of the aqueous humor and the membranes become thickened. Adhesion can occur between the anterior capsule and the iris or the edge of the wound, and later the iris and ciliary body are dragged by the contracting membrane. A chronic iridocyclitis is started in this manner, and may end in atrophy of the globe. Fortunately, in most instances, the inflammatory process subsides, allowing the institution of operative treatment. More formidable cases are those in which the iris is drawn into the corneal wound, and iris and capsule are bound into a cicatricial mass. The pupil is displaced and vision becomes much reduced. Such cases often terminate in iridocyclitis and detachment of the retina. In these cases, after all inflammatory symptoms have disappeared, Kuhnt's operation may be tried. In the *diagnosis* ordinary inspection shows a gray or whitish veil in the pupil behind the

iris, which is made more perceptible by the use of oblique illumination. A mydriatic is often useful in the determination of adhesions between the iris and membrane.

PATHOLOGY.—In a series of experimental operations made on rabbits Bates studied the changes which are attendant upon the development of post-operative cataract. The posterior capsule, which was not thickened, was generally wrinkled or thrown into folds. He did not find that secondary cataract was ever due to an opacity, thickening, or wrinkling of the lens-capsule, but attributes it to a development of new connective tissue. He found the first step to be an accumulation of coagulable fluid in the anterior chamber, which was followed by a deposition of fibrin. This was succeeded by new connective tissue. It is presumed that post-operative cataract in man is due to similar changes. Many authorities attribute post-operative cataract to the proliferation of epithelial cells in that portion of the anterior capsule which is not incised or removed at the time of the extraction of the lens.

TREATMENT.—The treatment of post-operative cataract, which is mechanical, will depend on the condition present. For ordinary membranous opacity the needle operation should be made with a Hay knife or a Knapp knife-needle. In case the iris and capsule are grossly adherent, and the pupil is drawn toward the site of the corneal wound, a more extensive operation, such as de Wecker's or Kuhnt's procedure, becomes necessary. In all operations for post-operative cataract two principles should be observed: First, the operation should not be undertaken until all inflammatory signs have disappeared; and, second, the offending membranes should be cut, never torn, since tearing produces traction of the ciliary body, which often results in loss of the eye. Discission of simple cases is followed by glaucoma in 1 or 2 per cent. Fortunately, this form of glaucoma yields to iridectomy.

Secondary Cataract is that lenticular opacity which is dependent upon pathologic changes taking place in tissues which are adjacent to the lens. The most common example is that which is seen in the cortical changes following neglected iritis, in which an exudate uniting the iris and lens is deposited on the anterior capsule. The resulting opacity is called *accrete cataract.*

Secondary cataract is also caused by such conditions as iridocyclitis, cyclitis, chorioiditis, glaucoma, and intra-ocular tumors. In these diseases the lenticular disturbances are attributable to interference with the nutrition of the lens. In most cases treatment is useless. In the accrete type, if the tension of the eyeball becomes increased, operation is indicated.

Complicated Cataract.—This condition arises when any form of cataract has some disturbing complication, such as some anomaly, inflammatory exudate, or traumatic disturbance, which may interfere with the usual process of ripening or may militate against a successful operation. In some cases the pupil appears black, giving the condition known as ***black cataract.***

It is a rare form of lenticular opacity, which is difficult of diagnosis. There is a gradual loss of vision, although the ability to see to count fingers is often retained. As a rule, the fundus cannot be illuminated. Oblique illumination shows fine striæ and a nacreous lustre.

The pathology of black cataract is uncertain. While its color is supposed to be due to an infiltration of hematin into the opaque lens, both Collins's and McHardy's cases spectroscopically examined failed to show any blood-pigment. Its treatment does not differ from that of ordinary senile cataract. Because it has a large nucleus, the incision for the extraction of black cataract must be of ample size.

Diabetic Cataract, which is but one of the forms of consecutive cataract, is commonly attributed to the abstraction of water from the lens, this being due to the altered composition of the intra-ocular fluids. While this form of lenticular opacity is probably the best known of the ocular manifestations of diabetes, the statistics as to its frequency differ much among various observers. It may occur at any period of life, and is generally bilateral. In young subjects, the lens being soft, there is a rapid development of the condition. In the old, by reason of the hardness of the lens, the process is slower and is less characteristic. In diabetic subjects the epithelium of the iris is much thickened and is easily loosened during operative procedures. Hence, during an extraction, the aqueous humor may suddenly become brown in color, from detachment of iris-pigment. Under proper dietetic and hygienic treatment such cataracts in adults may remain immature for long periods of time. According to Nettleship, the opacities may gradually disappear with the subsidence of the general symptoms. Cataract may be the only ocular manifestation of the dyscrasia, though often there are retinal changes and vitreous opacities. Iritis is a complication to be expected after operation.

PROGNOSIS.—This is favorable.

TREATMENT.—This will depend upon the condition of the patient and the state of the lens. In early cases, with beginning lenticular opacity, temporary correction of errors of refraction, regulation of diet, and the employment of internal remedies will sometimes result in checking the process. In children discission should be employed; in adults extraction should be preferred. After a technically successful procedure the visual result may be unsatisfactory because of degenerative changes in other parts of the eyeball. Diabetic coma may ensue, producing death several weeks after operation.

OPERATIONS FOR CATARACT.

This part of the subject may be divided into those procedures for the removal of senile, congenital, juvenile, and traumatic cataract, those for post-operative cataract, and operations for ripening.

Preparation of the Patient.—This is important. A bath should be given the night before operation and the excretory tracts emptied. Any

abnormal condition, especially of the mucous membranes, should, if possible, be removed. The field of the operation should be made surgically clean. This can be done by thoroughly washing the forehead, eyelids, and adjacent parts with soap and water, followed by a solution of bichlorid of mercury (1 to 2000). The conjunctiva is to be washed with the bichlorid solution after the anesthetic has been used and immediately before the operation is begun. The excess of fluid is reduced by gauze. It is not

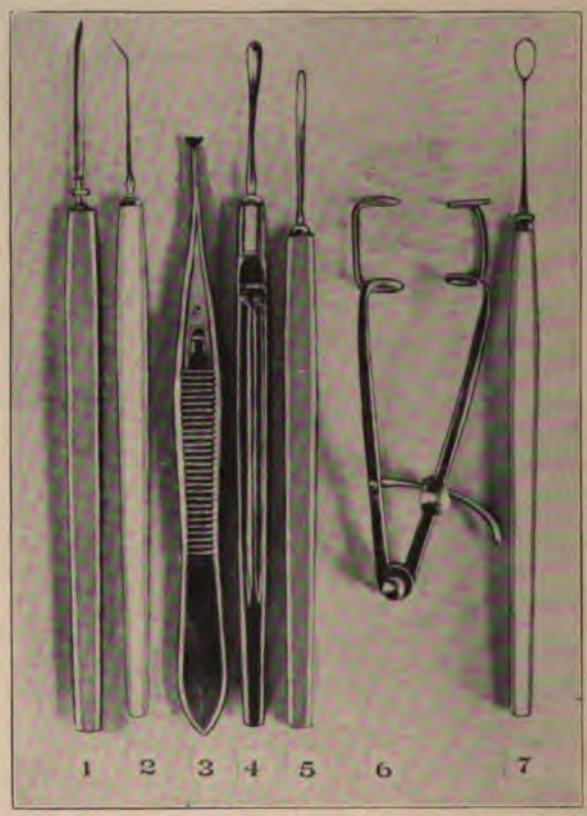


Fig. 306.—Instruments for extraction of cataract without iridectomy. (AUTHOR.)

1, Von Graefe knife. *2*, Angular cystitome. *3*, Fixation-forceps. *4*, Spoon. *5*, Spatula. *6*, Speculum. *7*, Loop.

advisable to permit the bichlorid solution to reach the interior of the eyeball, since it may produce keratitis. All instruments, gauze, dressings, and solutions are to be boiled before used. The operator and assistants should be surgically clean. The patient's hair should be covered with sterilized towels. It is presumed that the lacrimal apparatus and conjunctiva are healthy. If they are inflamed, it will be advisable not to operate until after inflammatory conditions have been cured.

Choice of an Anesthetic.—In all operations for traumatic cataract with rapid swelling of the lens and inflammatory symptoms, chloroform or ether is to be employed. In elderly persons, who have little self-control and are likely to behave badly, a general anesthetic often becomes necessary. In operations on infants and children the use of chloroform is advisable. For the majority of patients cocain anesthesia will suffice. Although many surgeons employ a weak (4-per-cent. strength) solution, the author uses a solution of 10-per-cent. strength. Weak solutions do not abolish the sensitiveness of the iris.

In patients in whom confidence can be placed, only a local anesthetic

Fig. 307.—Incision in cataract extraction. (AUTHOR.)

(Original drawing by DR. R. W. MILLS.)

is needed. For this purpose the operator can choose between a 10-per-cent. strength solution of cocain and a 1-per-cent. strength solution of holocain. It is perhaps unnecessary to add that the glass part of the eye-dropper must have been boiled and the solution is not to come in contact with the rubber bulb.

Extraction of Senile Cataract.—Extraction can be simple—that is, without iridectomy—or combined with iridectomy. The simple operation will be described first. The necessary instruments are: speculum, von Graefe knife, fixation-forceps (provided with a catch which can be easily opened), cystitome, spoon, spatula, and, as reserve instruments, if needed, iris-forceps, scissors, and lens-scoop. Besides a nurse, the surgeon should

have two trained assistants, one to attend to the handling of the instruments, the other to assist in the operation. The speculum is to be introduced into the conjunctival sac, the eye is to be held in one hand by fixation-forceps placed opposite the middle of the proposed incision, while the operator takes the knife in the other. He will stand either behind or in front of the patient during the operation. An upward section, which should include a little less than half the circumference of the cornea, is made in the apparent corneoscleral junction. The cystitome is used to cut the anterior capsule and the lens is gently delivered by means of the spoon. Any remaining cortex should be expressed by gentle movements of the spoon on the cornea directed from below upward. In difficult deliveries a spatula may be employed to press the posterior lip of the wound backward while the spoon is used in the manner mentioned. Any pieces may be removed by the blade of the spatula introduced into the anterior chamber. As a rule, efforts for the removal of cortex should be continued until the pupil appears black. The iris is replaced in the proper position by stroking with the spatula. The lips of the corneal wound are cleaned, the speculum is removed, and the lids are closed. A dressing of gauze covered with a light bandage is applied to both eyes. The eyes are allowed to remain undisturbed for forty-eight hours unless pain occurs, in which case the dressing is removed. Cleanliness must be maintained during the after-treatment. To prevent reaction atropin should be used on the third day. If much cortex has been unavoidably left, the medicine may be used in appropriate cases on the second day. If healing progresses favorably, the bandage can be left off and dark glasses substituted at the end of ten days. If all goes well the unoperated eye need be bandaged for only three or four days after operation. Temporary glasses are adjusted in four or five weeks.

In all cases the knife must be sharp and *the incision* should be in the same plane throughout. (The author prefers the knife made by Weiss, of London.) The puncture is made at a point one-half millimetre above the horizontal diameter of the cornea, commencing at the temporal side. The cutting is done by a gentle sawing movement, care being taken that the point of the knife does not touch the skin of the upper lid. At the finish of the incision a conjunctival flap can be made, or, as the author prefers, the knife can be turned slightly forward and the cut finished in the cornea. During this procedure the operator should be careful not to make undue pressure with the fixation-forceps. The incision of the capsule (known as cystotomy) is best made by a bent cystitome, whose point is passed into the anterior chamber and pupillary area; next it is passed under the iris and a wound is made in the periphery of the lens corresponding as nearly as possible to the general direction of the corneal incision. Some operators prefer to make a crucial cut in the pupillary area of the capsule or to excise a triangular segment of the same. During the use of the cystitome care should be taken that it does not injure the iris.

Delivery of the cataract requires a series of graded pressures. These should be applied in such a way as to cause the lower part of the lens to tilt directly backward, thus turning its upper segment forward into the corneal wound. This done, the delivery is accomplished by gentle, continuous pressure of the spoon.

EXTRACTION WITH IRIDECTOMY is accomplished in the following manner: After the corneal section is finished, the iris is gently drawn through the corneal wound and a small piece including the sphincter is excised. While the surgeon, holding the fixation-forceps in one hand, gently draws out the iris with the other, the cutting of the iris should be done by a competent assistant. The cystotomy, delivery, and dressing are made in the same manner as in simple extraction.

Accidents During and Following the Operation.—As regards *the incision,* the knife may be placed too far back, close to the dangerous ciliary zone, or too far forward in the cornea. Under these circumstances it is best to withdraw the instrument, treat the eye aseptically, and attempt the operation at a later period. If the knife is properly entered, but at the point of counter-puncture the conjunctiva is lifted up into a bleb by the aqueous humor, the section should be finished as if this had not happened. The knife may engage the iris either on the same side as that of the puncture or on the side of counter-puncture. If this happen, an attempt should be made to disentangle the blade and complete the section. In the event that the puncture and counter-puncture are properly made, but the iris falls over the knife, the section of the cornea should be finished without regard to the iris; except for the anterior chamber filling with blood and causing pain, this accident does no harm. Should the corneal section and cystotomy be made, but the lens fail to be delivered, this means, for example, either that the corneal wound is too small (a grave mistake) or that the capsule is tough and has not been properly cut. Under such circumstances the cystitome should be reintroduced and the capsule cut, followed by gentle attempts at delivery. If such measures fail, the incision, should it have been too small, must be enlarged by a scissors or a knife. Should the lens be dislocated, an attempt should be made to deliver it by means of a wire loop. The forcing of a cataract through too small a corneal opening is likely to produce disastrous inflammation, while prolonged attempts for delivery of a dislocated lens do damage. *Collapse of the cornea,* following the incision, is not infrequent. The operation should be continued as if this had not occurred. *The entrance of air into the anterior chamber* is of no importance. If it remains after the operation, it will soon disappear by absorption.

Spontaneous delivery of the lens immediately following the incision is a rare accident. Such a delivery is produced by spasmodic action of the extra-ocular muscles, and may be attended by loss of vitreous humor.

Loss of vitreous humor, if small, usually does no harm, and is followed generally by excellent vision, for the reason that the posterior cap-

sule has been torn. If a large amount is lost, the globe will collapse. When vitreous is expelled, the speculum should be removed at once. The protruding mass is to be snipped off with scissors and the eye closed. In cases where the vitreous humor protrudes so rapidly that the operator cannot use the scissors, the eye must be closed. A day or two later it may be found that the wound is approximated, no harm having been done, and small beads of vitreous will be seen floating in the conjunctival *cul-de-sac*. If so much of the humor has been lost that the eye collapses, the globe should be filled with normal salt solution. This procedure may save the eye.

PROLAPSE OF THE IRIS, coming immediately after a simple extraction, calls for an immediate iridectomy. If the prolapse occurs later and is noticed within forty-eight hours, the patient should be placed under the influence of chloroform and an iridectomy made. If noticed after strong

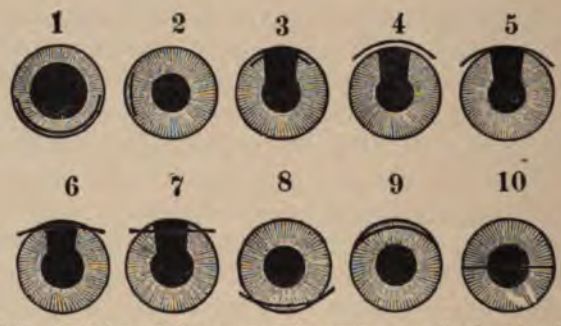


Fig. 308.—Incision of the cornea in various methods of cataract extraction. (NIMIER and DESPAGNET.)

1, Daviel's incision. *2*, Jaeger's linear extraction. *3*, Critchett's linear extraction. *4*, Von Graefe's modified linear operation. *5*, De Wecker's incision. *6*, Critchett's. *7*, Warmolont's. *8*, Liebreich's. *9*, Lebrun's. *10*, Küchler's incision.

adhesions to the corneal wound have formed, it should be let alone. If the iris, after replacement during the extraction, shows a tendency to draw toward the incision, it should be replaced, making the pupil central, and a drop of eserin or arecolin should be instilled into the conjunctival sac. Regardless of this indication some operators use miotics in every case of simple extraction.

During the operation should a *hemorrhage* come from the cut iris or conjunctiva, it is of no moment. It may so obscure the capsule that it cannot be seen during the performance of the cystotomy. Expulsive hemorrhage from the depths of the eye, causing pain, and coming on during or shortly after a cataract operation, is a serious accident. In such a case oozing of blood from the dressings will be noticed. Such eyes generally require enucleation. *Shock and delirium* are rare post-operative incidents in cataract patients. They should receive appropriate treatment (hypodermic injections of morphin, gr. $^1/_6$, or of hyoscin, gr. $^1/_{100}$). *Epileptic*

seizure, occurring during an extraction, is an unusual accident which is mentioned by Power.

Peculiarities in the Healing Process.—Generally a few hours after the extraction there is a little burning or smarting pain, which rarely calls for an anodyne. Severe pain coming on at any time within four or five days after operation will be due to hemorrhage, iritis, herpes, gout, or suppuration, etc.

If HEMORRHAGE appears late and is not profuse, a hypodermic injection of morphin should be given, the patient should be placed in the upright position, and the wound should be inspected. Some authorities advocate the opening of the corneal wound with removal of the blood by flushing the anterior chamber with a bichlorid solution. This advice is of questionable value. The careful cleansing of the conjunctival sac and of the lips of the wound, with full asepsis, may prevent suppuration and save a sightless globe. As regards that rare form of hemorrhage coming on after closure of the corneal wound, prophylactic treatment, with later needling, should be employed.

SUPPURATION IN THE CORNEAL WOUND.—Since the advent of clean surgery this complication is fortunately very rare. At the first dressing (some twenty-four to forty-eight hours after extraction) the color of the discharges on the dressing should be particularly noticed. If they be slight and of dark color, the eye may be said to be doing well. In such cases the eyelids need not be opened, unless it be thought best to begin the use of atropin early, as should be done where much cortex has been left behind. If the discharge be profuse, particularly if yellowish, and the upper lid is swollen, the lids should be opened and the wound inspected. If the edges of the wound are yellow, treatment to check the suppurative process must be promptly undertaken. The conjunctiva is to be washed with a warm bichlorid solution (1 to 4000), the edges of the wound are to be carefully cleansed with a stronger solution (1 to 400) of the same material, and atropin (1 per cent.) freely used. This plan of treatment is to be repeated twice daily. The employment of subconjunctival injections is of value in suppuration. For this purpose Darier recommends the following solution: Cyanid of mercury, 1; acoin, 10; normal salt solution, 1000. A few drops of this solution are to be injected beneath the conjunctiva three times a day. The acoin is added to render the injection painless. The injection should be made through an iridized platinum needle attached to a clean syringe. Morphin is employed to control pain; salines for the bowels, with supportive treatment, are indicated. In elderly, debilitated subjects the salines should be used sparingly, and quinin and milk punch are appropriate. If in twenty-four hours' time the eye is doing well, the same line of treatment should be continued; if the suppurative process extends, it will be necessary to chloroform the patient, cleanse the wound, and irrigate the anterior chamber with a solution of warm boric acid. The application of the electrocautery to the sloughing

area becomes necessary. The atropin should be continued. The placing of an iodoform disc in the anterior chamber has been proposed. Some authorities employ mercury freely by the mouth, or by inunction, or both (Schirmer, Herbert). Suppuration occurs at any time of the year. It is due to many causes, such as lack of cleanliness, rough handling of the eye, lacrimal disease, conjunctivitis, nasal disease, influenza, or lack of proper nutrition in the cornea. It usually occurs within the first three days; rarely after the fifth day. Pain is the danger-signal; the concomitant symptoms are chemosis, edema of the eyelids, turbidity of the aqueous humor, haziness of the cornea, and a profuse discharge of pus. It may end in panophthalmitis, or, in milder cases, the iris may be drawn toward the wound, the pupil be closed, and the eyeball be deformed by a cicatrix following the slough. In this latter type of cases an iridectomy is indicated after the eye becomes quiet. With careful attention to asepsis and skillful operating the condition will rarely happen.

Iritis is a frequent complication. Though usually occurring on the fourth, fifth, or ninth day, it may appear at any time within two weeks after an extraction. It may be due to rough manipulation during the extraction, or to an improper toilet of the wound. If any cortex has been left behind, the early use of atropin may be useful. If iritis occurs, the medicine may limit the degree of inflammation. A 1-per-cent. strength atropin solution should be used several times daily. If the attack should be severe, a solution twice this strength should be employed. The repeated application of cloths wrung in hot water are beneficial, or, if desired, dry heat can be applied by means of a Japanese hot box. In these cases it is advisable to dispense with all dressings, since they soon become wet and act as poultices. Dark glasses should be employed to protect the eyes.

Keratitis is also observed after cataract operation. Formerly, when bichlorid solution was permitted to enter the anterior chamber, a keratitis, presenting a milky-white appearance in the posterior part of the cornea and materially reducing vision, was often observed. The striped form of keratitis is a common condition after cataract operations. In this variety of keratitis numerous striæ pass downward from the wound. The condition disappears in a few days without treatment. The formation of vesicles in the cornea is a rare complication; it is supposed to be due to lymphstasis. It usually disappears in about two weeks' time.

Cyclitis may occur in eight or ten days' time in a case that has done well. In this form of complication a zone of circumcorneal injection will be noticed. The pupil may be round and the iris movable. Vision may be good. Pain, principally at night, may ensue. The capsule becomes thickened and vision is reduced. The process may pass away without leaving any bad results, or it may increase and terminate in glaucoma. The treatment consists in the local use of atropin, with the general employment of anodynes and sodium salicylate. Tension must be observed daily. If the eye becomes glaucomatous, an iridectomy should be made.

SPONGY EXUDATION INTO THE ANTERIOR CHAMBER may occur after extraction. It begins with pain and swelling of the lids and conjunctiva. The pupil becomes contracted and there is a turbid exudate like a cobweb situated in the anterior chamber. The condition disappears in about a week without treatment.

TARDY CLOSURE OF THE WOUND is generally due to the presence of foreign bodies, such as the iris, pieces of vitreous, or tags of capsule in the wound. Rarely it is caused by blepharospasm and spasmodic entropion of the lower lid. The anterior chamber may remain open for two or three weeks. The condition rarely leads to suppuration, to prolapse of iris, to infection, or to corneal opacity. Proper treatment consists in the application of compressive bandages. If complications occur, they must receive appropriate treatment.

Fig. 309.—Cystoid cicatrix lined by atrophied iris-tissue, in an eye in which glaucoma followed cataract extraction. (TREACHER COLLINS.)

1, Tip of the iris. *2*, Ciliary body. *3*, The bulging cicatrix lined by atrophied iris-tissue. *4*, Sclera. *5*, Cornea.

SPASTIC ENTROPION OF THE LOWER LID occasionally is seen in old persons. In such cases the bandage should be dispensed with; the eyelid may be held in position with adhesive strips.

FILTRATION EDEMA, from the subconjunctival escape of aqueous humor, may occur after the use of a large conjunctival flap. Such an edema is paler than the inflammatory variety, and is likely to gravitate to the lowest part of the conjunctiva. Aside from the use of the bandage, it does not require treatment.

ACUTE DERMATITIS, involving the eyelids and adjacent skin, has been observed by Herbert. It was attributed to a too free use of a solution of perchlorid of mercury, probably aided by bandaging. The use of a soothing ointment will be advisable.

CYSTOID CICATRIX.—A cystoid scar may appear after any form of trauma which opens the anterior part of the eyeball. It occasionally occurs

as a vesicle bulging from the site of the corneal wound after cataract extraction. It appears as a semitransparent vesicle. It may rupture at intervals and deposit aqueous humor beneath the conjunctiva. It is probably dependent upon the presence of a piece of capsular material that is caught in the wound, or, more often, is due to an incarceration of a part of the iris-tissue (either the sphincter or a surface attachment). Treacher Collins says that such cicatrices are lined by more or less atrophied iris-tissue.

When a fold of iris becomes fixed between the lips of a corneal wound the iris unites with the sclerocorneal tissue. The conjunctiva heals over it, and later, in response to intra-ocular pressure, this spot of least resistance expands. Normally the iris is impermeable to fluids, but the expanded portion becomes atrophic. Breaks appear in the pigmentary layer, which is probably the most resisting to fluids. A thin, bulging cicatrix, lined by atrophic iris-tissue, is thus formed. Any excess of intra-ocular fluid breaks through and passes into the subconjunctival tissue. Such a scar produces irregular astigmatism. Eyes affected with cystoid scars are likely to be lost from such conditions as iritis, iridochorioiditis, or iridocyclitis. Persistent cystoid cicatrix may be treated by dissecting a flap of adjacent conjunctiva, finding the minute fistula, and cauterizing it with the small point of the electric cautery.

Secondary Glaucoma.—This is a rare condition after cataract extraction, more frequently following discission. After extraction with iridectomy, it arises from adhesion of the pillars of the iris coloboma to the cicatrix and the lens-capsule, thus interfering with the drainage of the eye. Appearing several months after a successful cataract extraction, glaucoma may be due to the development of an epithelial implantation cyst in the anterior chamber.

Sympathetic Ophthalmitis is a rare condition appearing once in about eight hundred cases of extraction.

Choice of Cataract Extractions.—Extraction with iridectomy is safer for the patient in the hands of the majority of operators, the simple extraction requiring more skill. Those who operate only occasionally should make an iridectomy. Iridectomy is indicated if tension is increased, if the cataract is unripe or very large, if the patient is badly behaved, or if the iris is rigid: *i.e.*, does not react readily to a mydriatic. In hospital practice, where the lower classes congregate, it is safer to make an iridectomy. In private practice, among more intelligent persons, simple extraction can be made. The great advantage of the simple operation is the retention of a movable iris. The great disadvantage is the frequent occurrence of prolapse of the iris. This can often be prevented by the instillation of eserin or arecolin immediately after the delivery of the lens, or by an hypodermic injection of morphin, as advocated by Eugene Smith, of Detroit. The making of an iridectomy does not insure the eye against prolapse of the iris, for often the edges of the coloboma become attached to the lips of the

corneal wound. The question is an individual one which each operator must decide for himself. As shown by numerous statistics, the results as regards vision are practically the same.

Preliminary Iridectomy is always to be advised in case the patient has only one eye, or if complications exist or are to be expected. It is also to be advised in the extraction of lenses whose maturity is in doubt. A small portion of the upper segment of the iris should be excised through a corneal incision made preferably with a narrow keratome. Care should be taken to include the sphincter in the excised piece of iris. The extraction can be undertaken four or five weeks later.

Should Both Eyes be Operated on at the Same Sitting?—At the present day, when transportation and clinical facilities are so well developed, it is generally unnecessary to operate on both eyes at the same sitting. In charity practice the double extraction may be necessary. The consensus of opinion among modern ophthalmologists is in favor of operating on only one eye at the first sitting. Generally the extraction of the second cataract may be undertaken several weeks or months later. In a patient whose lenses are of about equal maturity, and who resides far from skilled ophthalmologists and will be unable to return, the surgeon should not hesitate to make the double operation.

Other Methods of Extraction.—Many methods of extraction have been advocated. Only those will be considered which are thought to be of importance.

Extraction with the Kalt Suture is a method of great value in critical cases. The suture is to be introduced before the corneal section is made and is to be tied after the lens has been delivered and the toilet of the eye completed. A short, sharp needle, curved on a short radius and without a cutting edge, is to be passed into the true corneal tissue, but not into the anterior chamber, about two and one-half millimetres from the limbus. The exit is to be made near the limbus. The needle is then to be passed into the episcleral tissue behind the limbus, leaving a loop of thread two inches long between the corneal and scleral portions. The incision having been made and the lens delivered, the toilet is to be finished and the suture tied. Risley, of Philadelphia, employs this method in cases where loss of vitreous is expected or the iris is tremulous, or the lens dislocated, requiring the use of the loop. Often by this means protruding vitreous can be pressed back into the eye and iris-prolapse can be prevented. In complicated cases, particularly if the other eye has been lost, the suture may be employed. It is to be drawn tightly, and removed on the fourth or fifth day. Although Kalt employs it as a routine method, the corneal suture has found favor with American ophthalmologists only in some complicated cases.

Extraction of the Lens in its Capsule would appear to be the ideal operation, since all tissue causing opacity is removed at one sitting. After making the corneal cut and iridectomy, the operator passes a scoop

behind the lens and lifts lens and capsule out together. The frequent occurrence of loss of vitreous humor has led to the abandonment of this operation as a general procedure. It is now employed for the removal of hypermature, tremulous, and dislocated cataracts and those presenting a calcareous deposit. Often in these cases the capsule is tough and the suspensory ligament is relaxed. Although the name of Pagenstecher has been frequently associated with it, the method originated with Richter and Beer.

THE SUCTION OPERATION, in which the fluid lens is drawn by suction through a tube introduced into the anterior chamber, is dangerous and unnecessary.

LINEAR EXTRACTION is open to the same criticism. In any case in which either of these operations seems indicated, the surgeon will do well to make a broad opening with the von Graefe knife and deliver the lens-substance as in the procedure advocated for traumatic cataract. Efforts to force *débris* through the linear opening may end in destructive intra-ocular inflammation.

IMMEDIATE CAPSULOTOMY.—So frequently does the capsule become thickened after a successful cataract extraction that some operators have attempted to make an opening in the posterior capsule immediately after the delivery of the lens. In the hands of a skillful operator it may be the ideal procedure. After extracting the lens in the ordinary way and removing all cortex, the operator introduces the point of a curved gold-enameled hook, made of malleable steel, which is passed on the flat into the anterior chamber behind the lower pupillary margin. The instrument is then turned backward, hooked into the capsule, drawn gently to the mouth of the incision, rotated on the flat again, and withdrawn. This procedure tears the capsule and permits the vitreous humor to come forward. Fox states that in patients in whom he performed this operation 15 per cent. required needling or capsulotomy with scissors, while of cases in which it was not performed 75 per cent. required later operations. The author has tried this operation and is not favorably impressed with it. Recovery is slow and iridocyclitis is likely to be a complication.

VARIETIES IN THE CAPSULOTOMY.—Some surgeons remove a piece of the anterior capsule with Mathieu's forceps, an instrument which has teeth on its lower surface. Eugene Smith, of Detroit, has long followed this method, and has devised a capsule-forceps. If the capsule be thin, a central piece is thus removed, leaving a black pupil; if thick, the capsule and lens may be dragged out together. An old operation, which is preferred by few modern ophthalmologists, is incision of the capsule with the cataract-knife while the instrument is being passed through the anterior chamber. There are several objections to this method of capsulotomy: (1) the incision in the capsule may be too small, (2) aqueous humor may escape before the counterpuncture is made, and (3) in the case of a Morgagnian cataract the field may be obscured by the opaque fluid.

EXTRACTION WITHOUT THE SPECULUM is preferred by some operators, who steady the globe with the fingers placed on the intervening lids, or the lids may be separated by means of retractors. By this procedure they hope to lessen the danger of prolapse of vitreous humor. If the patient is ill behaved, or the eye is deeply set in the orbit, the extraction should be made without the speculum. The lids are separated by the fingers of an assistant and the globe is fixed with forceps held by the operator.

It may be advisable to use the speculum only while making the corneal section, removal of the instrument becoming necessary if the patient becomes restless and squeezes the lids. An intelligent assistant can counteract the patient's effort by lifting the speculum away from the globe, thus preventing pressure on the eyeball.

DELIVERY BY PRESSURE OF THE FINGERS AGAINST THE GLOBE has been advocated, but is to be condemned because of the danger of producing infection. Instruments can be boiled; the fingers cannot be so treated.

WENZEL'S METHOD, which consists in the use of corneal and iris flaps, is of value in cataract with total posterior synechia. The knife, passed through cornea, iris, lens-capsule, and possibly through the lens, is made to emerge on the opposite side of the cornea. The tissues are then cut, a piece of the iris-capsule flap is to be excised with de Wecker's *pince-ciseaux,* and the lens delivered. The operation is of value in those desperate cases in which the whole posterior layer of the iris is adherent to the capsule. The loop, a sharp hook, or forceps may be required for the delivery of the lens. Loss of some of the vitreous humor is to be expected.

TYRRELL'S DRILLING OPERATION, which is sometimes of value in the treatment of cataract with iritic adhesions following sympathetic ophthalmitis, will be mentioned in Chapter XVIII.

DILATION OF THE PUPIL BEFORE EXTRACTION is practiced by some operators, particularly by the English surgeons in India.

Extraction of Immature Cataract.—While most surgeons delay the extraction until the lens has become ripe, and others approve of artificial ripening, there are those who favor extraction of immature lenses in persons about or above the age of sixty years. One of the strongest advocates of this practice is Schweigger, who has said: "At sixty years of age the lens is hardened in its entirety, whether cataractous or not, and whether the cataract is complete or not. The cataract and the induration are phenomena absolutely different; the one is pathologic, the other physiologic. Thus, in persons of sixty years who have lost the faculty of accommodation, a cataract even incompletely developed may be removed without the opacities being reproduced, for it is certain that the lens can be removed entirely." If, under these circumstances, extraction is undertaken, an iridectomy should be made and special attention should be given to the removal of cortical substance. If much cortex remains, iritis will be sure to develop. The use of atropin immediately after extraction will be advisable. It should be used until all corneoscleral redness has disappeared.

Some surgeons, of large experience in the extraction of immature cataract, advocate irrigation of the anterior chamber with a warm saline solution, immediately following the delivery of the lens. McKeown operates in this manner: After the corneal section and a small iridectomy have been made, a fine hollow needle is introduced inside the capsule of the lens. Then a few drops of the solution are injected, by siphonic action, beneath the capsule. The cystotomy is then made and the lens-nucleus delivered. The cortical substance is then to be removed by irrigation.

Scleronyxis, or Keratonyxis (Dislocation of the Lens; "Couching").—This ancient operation, which has practically become obsolete, consists in depressing the lens into the vitreous body by means of a cataract-needle passed through the cornea or sclera. The operation as a routine procedure justly has been abandoned by reason of its many sequelæ. While the immediate effect of the dislocation of a cataractous lens is most brilliant, the ultimate result is disastrous, the majority of patients becoming blind in a few years from detachment of the retina, iridocyclitis, glaucoma, chorioiditis, hyalitis, etc.

Recently Powers, of London, and Suker, of Chicago, have revived the operation, which is to be employed only in carefully selected cases. Suker considers the operation justifiable in the following conditions: Cataract in the insane, epileptic, hemophilic; in cases with tremulous iris or fluid vitreous humor without chorioiditis or retinitis; in incurable dacryocystoblennorrhea, or conjunctivitis; in extreme old age, with its attending infirmities; in patients with incurable bronchitis and cough; in cases in which, one eye having been operated and lost by suppuration, the indications point to danger of the same disaster to the other eye; in shrunken and secondary cataracts; and in posterior dislocations with tremulous iris. While the author believes that this list is too long, he can conceive of cases in which depression might be justified and extraction not warranted. Extreme age alone is not a barrier to successful extraction and mild forms of insanity do not prohibit successful operations. In persistent dacryocystoblennorrhea excision of the diseased area, followed a month later by extraction of the lens, should be preferred. Nor can the failure of an extraction on the first eye be justly advanced as an argument for couching. The operation of couching, however, may be considered justifiable in violently insane subjects; in those epileptics who are subject to daily seizures, and in those cataract patients of advanced age who, being near death, wish to see the faces of their loved ones once again.

The operation of couching requires a speculum, a fixation-forceps, and a cataract-needle. The pupil having been dilated, the needle is passed through the cornea to the upper border of the pupil and placed against the lens. Then gradually increasing pressure is used to rupture the zonula and force the cataract backward into the vitreous. If the needle becomes engaged in the lens it can be dislodged by slightly rotating the instrument on its axis. It can then be withdrawn without danger of the lens following

into the anterior chamber. The eye should be bandaged and kept at rest for a few days. Atropin should be used in the after-treatment.

Operations for Juvenile Cataract.—In the complete opacity of congenital and juvenile cataract discission is the proper operation. If a large part of the lens is clear a visual iridectomy should first be tried. Discission, or the needle operation, requires a speculum, fixation-forceps, and two cataract-needles. The pupil having been dilated and the patient being under a local or general anesthetic, the same preparation of patient, operator, and instruments is to be employed as in extraction. The speculum having been introduced, the operator, holding the globe with the forceps in the left hand, with the right passes a cataract-needle through the cornea into the lens-capsule, making a crucial incision. The instruments are then to be withdrawn, more atropin is applied, the conjunctiva is washed with a bichlorid solution, both eyes are bandaged, and the patient is placed in

Fig. 310.—Depression of cataract. (AGATZ.)

bed. Atropin is to be continued until all signs of irritation have disappeared. In favorable cases both eyes should be covered for four or five days. Care must be taken not to dislocate the lens by the employment of too much force, nor to open the capsule too extensively. Pain and iritis are to be overcome by atropin and heat applied as previously explained. If there is rapid swelling of the lens with the advent of increased tension, the cornea should be opened with the von Graefe knife and the lens delivered as in the operation for traumatic cataract. Dunn claims that if the aqueous humor is permitted to drain away before withdrawing the needle, thus producing temporary minus tension, there will be no danger of glaucomatous symptoms following discission. The object of the needle operation is to bring the aqueous humor into contact with the lens. Solution of lens-substance then occurs and absorption follows. It is far safer to repeat the operation several times at intervals of a month or more than

to make too extensive an opening at one time. In the later stages, in cases where the posterior capsule is tough, two needles should be used, as shown in Fig. 311, thus avoiding traction on the ciliary body. While generally the needle operation gives excellent results, serious inflammation and even loss of an eye may happen to the most careful surgeon.

At what age should discission give way to extraction? In general terms it may be said that discission is rarely admissible after the twentieth year. In high myopia the needle operation is sometimes employed to cause absorption of a clear lens. This subject will be discussed in the chapter on "Refraction."

Operations for Traumatic Cataract.—Where the lens is injured and swells rapidly, causing increased tension, it should be extracted by the method to be described. For years it has been the practice to employ the linear operation. Performed for the purpose of relieving undue tension

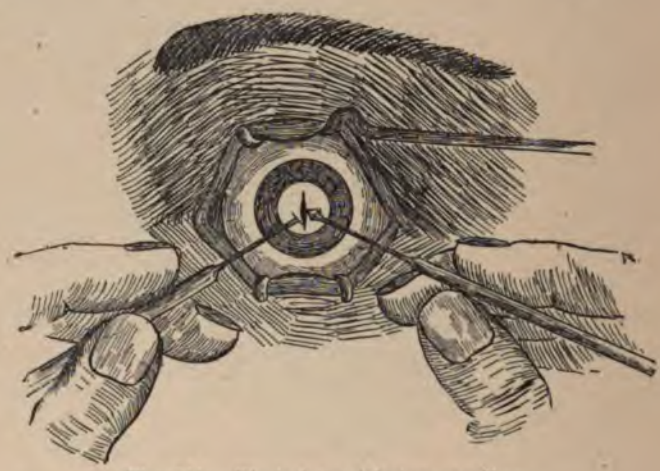


Fig. 311.—Discission with two needles.

and evacuating lenticular fragments, the nature of the operation is such as to diminish the first only temporarily and defeat the second frequently. The site of the incision in the linear operation precludes the possibility of removing all fragments of the swollen lens. The oblique course of the wound, shown in Fig. 312, renders its patency impossible while favoring its closure. Furthermore, the incision made with the keratome is often too short. Flap wounds of the cornea gape more than linear ones, the tendency to gape depending upon whether the wound traverses the tissues perpendicularly or obliquely. A perpendicular section can be made easier, safer, and better with the von Graefe knife than with a keratome. If the instrument passes obliquely through the cornea, the lips of the wound close like a valve. The closure is due to intra-ocular pressure. This force, in the linear operation (see Fig. 312), acts as strongly on the posterior lip *(3)* as the anterior *(2)*. The wound must be made to gape before

lens-substance can be expressed. Gaping of the wound can be produced, not by pressure opposite the site of the incision, but by force applied peripherally to the wound (at 4 in Fig. 312). Such an operation is unscientific. In place of it, the author for several years has followed this procedure: The patient being under a general anesthetic, the pupil having been dilated, a von Graefe knife is passed into and out of the cornea, as in the extraction of senile cataract. The incision includes nearly one-half the circumference of the cornea. Such a wound will permit the removal of all swollen lens-substance, and frequently it will obviate the necessity of an iridectomy. Attention to asepsis is imperative. Atropin drops are to be continued for ten days.

Procedures for Post-operative Cataract.—Following a successful extraction the lens-capsule may become thickened. In the majority of cases this occurs at a period varying from a few months to several years. The result is a great diminution of vision. The remedy is to remove a central piece of the offending membrane, or to make an opening in it sufficiently large for visual purposes. The obstruction to vision may come from a

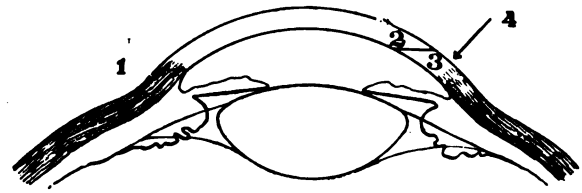


Fig. 312.—Site of incision for traumatic cataract. (AUTHOR.)

1, Site of incision in the author's method of extraction in traumatic cataract. *2*, *3*, Site of incision in the linear operation.

closure of the opening which was made in the anterior capsule at the time of operation. Proliferation of the epithelial cells of the capsule follows, and thus a tough, gray membrane is formed. In another series of cases in which peripheral capsulotomy has been performed, both anterior and posterior capsules are thickened. In still another type adhesions exist between the iris and capsule, and subsequent contraction of the mass exerts injurious traction on the ciliary body. A grave form of opacity is that in which the pupil has been drawn into the corneal wound. The iris is then placed on the stretch, and a solid mass, composed of iris, capsule, and exudate, obstructs the view. The simple form of obstruction shows a black pupil by ordinary inspection, but oblique illumination reveals a delicate, wavy, gray membrane in the pupillary area. In this condition the following operation should be made: After dilating the pupil, preparing the parts as for an extraction, and anesthetizing the eye, the operator passes two very sharp needles through the cornea, as in Fig. 311. The points of the needles should be made to pass through the same opening in the capsule. Then, the handles being brought together, the needle points are separated, each needle forming a support for the other, so that traction

will not be made upon the ciliary body. The capsule should be cut, not torn. The needles are to be withdrawn, atropin used for ten days, and glasses prescribed after all signs of irritation have disappeared. If intraocular tension rises, a paracentesis of the anterior chamber should be made and the atropin should be replaced by a 2-grain strength solution of pilocarpin used thrice daily. If the tension is not reduced, or again rises, a broad iridectomy should be made at once. This will reduce the tension. This form of glaucoma occurs in 1 or 2 per cent. of needle operations. Another danger is infection, which occasionally destroys an eye after this procedure.

Instead of employing the needle operation, Callan pierces the cornea and capsule with a long, narrow keratome. After withdrawing this instrument de Wecker's scissors is to be passed into the eye, one blade being in front, the other behind the capsule, which is then to be cut. Thus a T-shaped aperture is made. If the capsule is not unduly tough, a clear pupillary area can be obtained by the use alone of the long angular keratome. This is passed through the cornea and capsule, and, while still *in situ,* is made to move from side to side, thus producing a larger capsular opening. This procedure requires a very sharp instrument. It is not

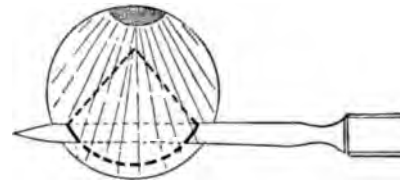


Fig. 313.—Operation for post-operative cataract. (KUHNT.)

applicable to thick, tough capsules, and can be employed only with the pupil dilated. As in other similar procedures, atropin is to be used in the after-treatment.

In those serious cases of post-operative cataract, where the iris has been drawn into the corneal wound and a dense membrane fills the pupillary area, a more radical operation will be necessary. The best procedure is to make a large corneal opening and with de Wecker's scissors cut out as large a section of the membrane as possible, always avoiding traction on the ciliary body. Among numerous operations suggested for this purpose, that of Kuhnt is one of the most valuable. A cataract-knife passed through the cornea, iris, and membrane is brought out on the opposite side, as shown in Fig. 313. Then with the de Wecker scissors a large part of the obstruction is to be excised.

Artificial Ripening of Cataract.—Some cases of binocular cataract may ripen very slowly, necessitating a long period of waiting, which may be injurious to those who are wage-earners or those who have business interests. In such cases, the other parts of the eye being in good condition, there should not be any hesitation to ripen the lens. For the performance of this procedure the pupil should be dilated by a mydriatic.

The anterior chamber is to be opened with a keratome and the lens is to be triturated by means of a small spatula. In a few weeks' time the artificially ripened lens can be extracted. This operation is not of value in posterior polar cataract, in slowly sclerosing lenses with few striæ, and in the slowly forming cataract of myopia.

OPERATIONS FOR ARTIFICIAL RIPENING.—Numerous procedures have been devised for the artificial ripening of cataract. They can be tabulated as follows:—

1. Simple division of the anterior capsule.
2. Division combined with iridectomy: Mooren's operation.
3. Division and external massage.
4. Iridectomy and external massage: Förster's operation.
5. Iridectomy and internal massage directly on the anterior capsule: Bettman's operation.
6. Simple paracentesis with indirect massage: White's operation.
7. Paracentesis with direct massage.
8. Injection by the Pravaz syringe of fluid beneath the anterior capsule: Jocqs's method.

The question arises: Which one of these is the best operation? Before performing any of these procedures the operator should know that the zonula is intact, the tension not below normal, and the pupil dilatable.

1. Simple division of the anterior capsule is condemned by all authorities on account of the inflammatory changes which follow a rapidly swelling lens. These are not only iritis, but also panophthalmitis. The most experienced ophthalmic surgeon cannot always tell when a wound of the lens will be followed by a dangerous reaction. The opening in the capsule may be too small. The surgeon may lacerate the fibres too extensively and cause the lens to swell too rapidly. Sometimes the result is entirely negative, the operation causing a plug of cortex to be squeezed out of the capsular wound, which afterward closes. The same objections obtain in the case of (2) division combined with iridectomy and (3) division with external massage.

4. Iridectomy and external massage is the operation formerly most frequently performed, and is to be preferred to any procedure in which the capsule is divided. However, there are some objections to it. To those who prefer the simple extraction, the iridectomy would be a serious objection. Although the pupil is dilated by atropin before commencing Förster's operation, some fibres of the iris are likely to be pressed between the lens and cornea, thus causing iritis. Mittendorf has recorded the loss of an eye. It must be remembered that the cornea also is being triturated, and its epithelial coat may be rubbed off. Förster himself has called attention to the danger of injury to the cornea and iris, and to the possibility of producing a dislocation of the lens. The danger of dislocating the lens pertains as well to other operations for artificial ripening.

5. The operation of direct massage is the best procedure. It can be done with (5) or without (7) iridectomy. The trituration is made

directly upon the lens-capsule. The iris is not compressed; hence the danger of iritis is less than in Förster's operation. The percentage of successes will be found to be greater than in any operation where the massage is indirect.

6. White's operation, or paracentesis and external massage. The same objection (danger of compressing the iris) as that applied to Förster's operation also obtains here.

7. For the Bettman operation these instruments are needed: speculum, fixation-forceps, keratome, iris-forceps, scissors, and spatula. The spatula is made of aluminum. It is twenty-eight millimetres long and one millimetre wide. The cornea is to be opened by means of the keratome, the iridectomy made, and gentle massage applied by passing the spatula into the anterior chamber under the iris and on the anterior capsule. Ten or twelve movements will suffice. Atropin is to be continued for a week. Three or four weeks later the extraction can be made.

8. Jocqs's method is new, and has not been sufficiently tested to permit an opinion as to its value.

Refraction after Extraction.—Removal of the lens leaves the eye aphakic. To secure good vision, a convex lens must be placed in front of the eye. To this must be added a convex glass of about three dioptres' strength in order that the patient may read. To glasses both for far and near work there must be added a cylinder to correct the astigmatism remaining after the operation. This averages about three or four dioptres. The astigmatism following cataract extraction is greatest immediately after the extraction and progressively diminishes for six or eight weeks or longer. In some cases there is a further normal reduction in astigmatism, and glasses must again be prescribed at the end of six months. Here, as elsewhere, the estimation of the amount of astigmatism, and the determination of the axis at which the cylinder is to be placed, is much simplified by the use of the astigmometer.

Results of Cataract Operations.—Statistics compiled from the reports of skillful surgeons show that the result of cataract extraction is the restoration of a useful amount of vision in 95 per cent. of uncomplicated cases. Of the remaining cases, 3 per cent. will have perception of light; 2 per cent. will be totally blind. In tabulating their cases, ophthalmic surgeons consider vision of $^{20}/_{200}$, or better, to be a *good result.* Vision from $^{1}/_{200}$ to $^{18}/_{200}$ is classed as a *moderate result,* while cases possessing only perception of light are classed with the totally blind as *failures.*

Spontaneous Cure of Cataract.—Spontaneous disappearance of senile cataract occurs so rarely that the surgeon is not justified in mentioning it to a patient. Brettauer, in 1885, reported three cases of spontaneous absorption of senile cataract, with restoration of vision, in all of which he had removed a cataract from the fellow-eye. In these cases there was no sign or history of injury, operation, or disease. Pyle has classified the reported cases as follows:—

1. Cases of absorption after spontaneous rupture of the anterior or posterior capsule.

2. Cases of spontaneous dislocation of the cataractous lens.

3. Cases of intracapsular resorption of the opaque cortex and sinking of the nucleus below the axis of vision, after degenerative changes in Morgagnian cataract, without rupture of the capsule or dislocation of the lens.

4. Cases of complete spontaneous resorption of both nucleus and cortex without reported history of ruptured capsule, dislocation, or degeneration of the Morgagnian type.

5. Cases of spontaneous disappearance of incipient cataract without degenerative changes or marked difference in the refraction.

DISLOCATION OF THE LENS, ETC.

Acquired Dislocation of the Lens may occur from trauma or spontaneously. If from trauma, the other structures of the eye may be seriously injured simultaneously. The lens may be thrown backward into the vitreous body, or forward into the anterior chamber. It may pass through a corneoscleral rupture into a bed beneath the conjunctiva, or into the capsule of Tenon, or may be extruded entirely from the eye. At present only dislocation without rupture of the globe will be considered. Spontaneous dislocation occurs in cases in which a congenital defect or acquired disease has weakened the zonula of Zinn. Atrophy of the zonula may result from liquefaction of the vitreous, chorioiditis, high myopia, or detachment of the retina, and is not infrequent in hypermature cataract. The zonula being weakened, a slight disturbance, such as coughing, sneezing, or bending over, will suffice to cause a dislocation. The lens may completely leave its bed, luxation; or it may remain partly in place and be tilted forward or backward, subluxation. Disloçation into the vitreous humor is more frequent than into the anterior chamber. If the vitreous is softened, the lens can be seen bobbing about whenever the eye is moved. A misplaced lens becomes opaque, but in the vitreous it may retain its transparency for a long period. It acts as a foreign body and can cause serious trouble. The uveal tract becomes irritated, and such eyes often end in iridochorioiditis. If dislocated into the anterior chamber, the lens may block the drainage apparatus, causing pain, increase of tension, and destruction of vision. In subluxation the patient is likely to be myopic. The zonula having lost its power over the lens, the latter by its elasticity gains greater curvature, thereby increasing the refraction.

Diagnosis.—A partly dislocated lens, if clear, may cause monocular diplopia. When the lens is in the vitreous humor, the anterior chamber looks deep; the iris is tremulous, accommodative power is lost, the pupil usually is enlarged, and often the dislocated lens can be seen by the naked eye. To the ophthalmoscope the eye is markedly hypermetropic and the lens can be found as a dark, grayish body, constantly shifting its position. When in the anterior chamber, the lens, by ordinary illumination, appears of a golden or amber color much resembling a large drop of oil. It may

wander from the vitreous to the anterior chamber in response to the position of the eye. It may remain for weeks in the anterior chamber, setting up great inflammation, and suddenly be found in the vitreous.

PROGNOSIS.—An eye with its lens located in the anterior chamber, and without other lesions, offers a favorable prognosis. If glaucomatous symptoms have appeared, the outlook is not so favorable, although it is surprising how well some apparently desperate cases will do after removal of the irritating body. A dislocation into the vitreous, if recent, may offer a good prognosis, provided the surgeon can succeed in getting the lens into the anterior part of the eye, from which it can be extracted. An old dislocation, with a fluid vitreous humor, offers little encouragement.

TREATMENT.—The proper treatment is extraction. If the lens is in the anterior chamber, removal will be easy. The first step will be the anchorage of the lens. A local anesthetic is to be used and a speculum introduced. The lens is to be fixed by passing an ordinary straight saddler's needle, held in a Sand needle-holder, through the cornea. Then with a von Graefe knife the cornea is to be opened as in the ordinary

Fig. 314.—Dislocation of the lens into the anterior chamber. (VON AMMON.)

extraction. A wire loop is to be passed behind the lens, which is then delivered. The use of the bident is not necessary. The treatment is the same as that employed after a cataract operation. If the lens has caused great inflammation, or if the patient is a child, a general anesthetic will be necessary. It should be given with the patient placed face downward. Otherwise, when ready to operate, the surgeon may find that the lens has passed into the vitreous humor. It may be advisable to anchor the lens under holocain or cocain and deliver it under chloroform. If it is situated in the vitreous chamber, attempts to remove it are not justifiable unless its position can be changed. With the pupil dilated and the patient placed face downward, the lens may drop into the anterior chamber, in which event eserin should be instilled immediately, in the hope that the contracting iris will hold it in position until it can be anchored. In these cases, owing to the great danger of loss of vitreous humor, it will be advisable to make use of the Kalt suture. If an eye containing a lens located in the vitreous humor is blind and painful, an enucleation should be made to prevent the occurrence of sympathetic ophthalmitis.

Parasites in the Lens.—The parasites which have been found in the lens are filaria, monostoma, and distoma. The filaria, filaria lentis, is a species of which the female has been found several times in cataractous lenses. It measures from one to three millimetres in length. For obvious reasons it is found in hot climates, particularly on the west coast of Africa.

Spontaneous Extrusion of the Lens is of very rare occurrence. In a case reported by R. J. Hamilton, an infant of four months was brought to him with an apparent rupture of the corneoscleral margin. The mother stated that the lens had come out while she turned the child on her knee. A week later the night nurse reported that the other lens had been extruded, it being found on the pillow while the child was asleep. The child died the following day and post-mortem examination showed that the vitreous chamber was occupied by a white, opaque membrane which was attached posteriorly to the centre of the optic disc, from which it spread out in pear-shape and was attached at several points to the chorioid. In contracting it had separated the chorioid from the sclera. The contraction of this membrane was the *vis a tergo* which was sufficient to expel the lens through what is the weakest part of the eye in early life, viz.: the corneoscleral margin.

Ossification of the Lens has been observed by Panas and others.

Regeneration of the Lens.—The reproduction of the human lens after extraction has never been demonstrated, although Vrolik claimed to have seen "a transparent new formation" in the site of a crystalline lens which had been removed eleven years previously for cataract. Regeneration of the lens in some of the lower animals—as rabbits, dogs, and guinea-pigs—does occur. Recently Randolph has published the results of twenty experiments made on newts in which he obtained regeneration in six. Since "regeneration occurs even when the lens has been removed in capsule," he concludes that the new lens takes its origin from the iris, thus confirming the experiments of Wolff. Great activity of the capsular epithelium is often noticed after the extraction of cataract. Randolph asks: "May not this be an attempt at regeneration, an attempt which is so often successful in creatures lower in the animal series?"

Injuries of the Lens.—These include rupture of the zonula, dislocation, rupture of the anterior or posterior capsule, and opacity without visible lesion (concussion cataract).

Penetrating injuries without the retention of a foreign body are occasionally seen. Among the agents concerned with the production of such injuries are thorns, needles, and knife-blades. The injury may involve the zonula or the peripheral part of the lens, in which case the iris will conceal the wound. After perforation of the zonula near its lenticular attachment a stellate opacity of the posterior layers of the cortex is likely to occur. Often, in these cases, if the eye is not lost by infection, there will be complete opacity of the lens. In rare instances the wound in the capsule will heal and lenticular opacity will completely disappear. There

may be rapid swelling of lens-fibres and increased intra-ocular tension, which will necessitate an immediate extraction. In cases of less severity the use of atropin will be required.

Foreign Bodies in the Lens.—Not infrequently foreign bodies, such as pieces of iron, steel, or copper, will lodge in the lens. They may be visible or hidden, depending upon the depth to which they penetrate, upon the amount of lenticular swelling, and upon the time which has elapsed since the receipt of the injury. If hidden, they can be localized by means of the x-rays. Not infrequently it will be found that the foreign body has either passed through the lens into the vitreous, retina, chorioid, sclera, or orbital tissues, or has lodged in the ciliary region.

CHAPTER XIV.

DISEASES OF THE VITREOUS HUMOR.

Coloboma of the Vitreous Humor is a rare congenital anomaly which appears as a cleft in the under part of the vitreous body, extending in the most marked instances from the optic papilla to the ciliary region, while in lighter grades it occupies only the anterior or posterior part of the vitreous.

Persistent Hyaloid Artery (Canal of Cloquet).—An unusual anomaly is the extra-uterine existence of the hyaloid canal: a lymph-space in the axis of the vitreous body. The hyaloid artery, which during fetal life is an extension of the central retinal artery, running in the canal of Cloquet, generally disappears about the seventh or eighth month of intra-uterine life. When persistent, it forms a rare anomaly and runs forward in the

Fig. 315.—Coloboma of the vitreous humor. (ECKER.)

vitreous, sometimes reaching the posterior surface of the lens. It may be seen floating in the vitreous in one of several forms: as a strand attached either to the papilla or lens, as a vitreous vessel carrying blood, as a canal without a vessel, or as a cyst-like body. Its presence is not incompatible with normal vision, and the anomaly is chiefly of interest as a scientific curiosity. In a case recently examined by the author the canal was present in both eyes as a funnel-shaped, grayish, floating body. Traces of the hyaloid artery are also sometimes seen at the posterior pole of the lens, as a speck on the capsule, and at the optic disc as gray, connective-tissue tags. According to Mittendorf, these small opacities of the posterior capsule are non-progressive, do not interfere with vision, and are found in 2.3 per cent. of eyes.

Inflammation of the Vitreous Humor.—The term hyalitis is applied to inflammation of the vitreous body (and not to the hyaloid membrane), which presents two forms: one suppurative and the other connected with the formation of opacities. Strictly speaking, the changes which are about to be described are not distinct diseases, but are simply manifestations

of pathologic changes in the retina, ciliary body, chorioid, or the optic nerve, or are due to trauma. Since the nutrition of the vitreous depends on the vessels in the ciliary body and retina, it is apparent that lesions in these parts readily affect the vitreous.

Suppurative Hyalitis (Purulent Inflammation of the Vitreous) is part of the condition known as panophthalmitis. It is caused by trauma, particularly in connection with penetrating wounds with the lodgment of a foreign body; and occurs idiopathically in connection with metastatic chorioiditis following puerperal fever, exanthematous diseases, influenza, and low fevers. Recently it has been shown that idiopathic inflammation of the vitreous humor may occur without demonstrable changes in any other part of the eye. Suppurative hyalitis may also be caused by microbic invasion through old operative wounds, the inflammation appearing from a few months to several years after healing.

Fig. 316.—Canal of Cloquet, with cyst in the vitreous humor. (KOLLER.)

("Transactions of the American Ophthalmological Society," 1901.)

SYMPTOMS.—The patient usually gives the history of an antecedent iritis or iridocyclitis, and adhesions between the iris and lens-capsule can generally be demonstrated. If the cornea and lens are clear, pus can be seen in the vitreous as a circumscribed, yellowish, opaque mass, which ophthalmoscopically may simulate neuroepithelioma of the retina. To this condition the name pseudo-glioma is usually applied. In making a diagnosis it will be necessary to consider the history of the case, the age of the patient, and the appearance of the mass (see the diagnosis of neuroepithelioma of the retina in Chapter XV). Tension in suppurative hyalitis is usually diminished, but may be increased. Generally the diagnosis will not be difficult. Acute suppurative hyalitis producing a yellowish mass behind the lens, increased tension, and inflammatory symptoms may be mistaken for a neoplasm. The rapid course of the disease and the history of the case will usually serve to clear the diagnosis. When scleral perforation occurs with a discharge of pus, there will no longer be doubt as to the existence of suppurative hyalitis.

PROGNOSIS AND TREATMENT.—The prognosis in suppurative hyalitis is serious. The affected eye will be lost and may be a cause of sympathetic ophthalmitis. Shrinking of the globe occurs. Old, tender, cyclitic eyes should be removed. During the course of an exhausting disease the surgeon may notice vitreous opacities appearing as fine flakes. In such a case it is possible that vigorous supportive treatment may check the process and save the eye. The possibility of the occurrence of a vitreous complication in low fevers should not be forgotten by the physician. The treatment of suppurative hyalitis in the present state of our knowledge is purely symptomatic. Berry and others have advocated the intra-ocular injection of chlorin-water on experimental grounds, but such treatment is not to be sanctioned. The intra-ocular use of iodoform may be of value.

Opacities of the Vitreous Humor.—These are classified as fixed and floating, and may be acute or chronic. They may appear as fine, dust-like particles (particularly in syphilitic retinitis and chorioiditis) or may be threads, bands, dots, membranes, or projections. They vary much in size, shape, and color. The fixed opacities are generally fastened at two or more points.

ETIOLOGY.—Opacities of the vitreous result from many different conditions, among which are the following: Errors of refraction, particularly myopia of high degree; inflammation of the retina and uveal tract; traumatisms producing hemorrhages from the ciliary region or chorioid; depraved conditions of the general system from low fevers or other exhausting diseases, anemia, loss of sleep, gout, syphilis, menstrual disorders, constipation, portal congestion, malaria, the long-continued use of arsenic, etc. Large fixed opacities may result from trauma and may simulate neoplasms. In many instances opacities of the vitreous are present in persons in whom other affections are not demonstrable; this is particularly true of elderly persons. Under the name asteroid hyalitis Benson has described a condition in which small, light-colored spheres are found in the vitreous. It is probably a congenital condition.

SYMPTOMS.—The subjective symptoms of vitreous opacities depend on the number and size of the opaque spots. The patient sees them as black specks floating in the visual field (muscæ volitantes). They are particularly annoying when the patient is looking at a bright surface. Visual acuity may or may not be reduced. Reduction of vision, pain, and asthenopic symptoms will cause the surgeon to seek errors of refraction, associated ocular changes, or diseases of the general system.

Vitreous opacities can be seen by using a concave retinoscopy mirror of twenty-five centimetres' focus. Whether the spots are fixed or floating can be determined by having the patient move his eye while under examination. After movement the eye is to be held perfectly quiet. Fixed opacities will remain *in situ*, while floating ones will be seen to move, and their rate of movement gives an indication of the consistency of the vitreous. In a vitreous body of normal consistency the spots move slowly; in a fluid

vitreous the movement is rapid. Vitreous opacities, if floating, move in a direction opposite to the movement of the eye, while opacities in the lens and cornea move in the same direction as the ocular movement. This is because the latter opacities are fixed. By using a + 14 or 16 D. lens, by direct ophthalmoscopy, fixed vitreous opacities can be examined satisfactorily.

PROGNOSIS AND TREATMENT.—The prognosis in vitreous opacities depends on the cause. When resulting from syphilis the opacities readily clear up under proper treatment. If caused by hemorrhage, the clot may or may not be absorbed. In opacities caused by purulent chorioiditis the prognosis is grave, the eye ending in phthisis bulbi. Muscæ volitantes show no opacities to the ophthalmoscope, and are probably caused by shadows of the remains of embryonic tissue thrown on to the retina. They are exceedingly annoying, and often remain in spite of the correction of refraction errors and attention to the general health. They are not significant of serious lesions.

The treatment of opacities of the vitreous includes the removal of the cause, the regulation of the habits of the individual, and the exhibition of remedies for the purpose of producing absorption. Iodid of potassium, the various preparations of mercury, pilocarpin, and saline laxatives are all valuable measures. The use of the galvanic current, paracentesis of the anterior chamber, and the subconjunctival injection of a 5-per-cent. strength salt solution are among the measures which have been advocated. The intra-ocular injection of various mercurial salts, iodid of potassium, and carbolic acid has been proposed. This method of treatment should be condemned, as it is dangerous to the eye.

Hemorrhage into the Vitreous Humor.—This may occur spontaneously from rupture of a diseased vessel or as the direct result of trauma. Idiopathic hemorrhage occurs chiefly in chorioiditis, retinitis, high myopia, and in elderly persons with atheromatous arteries. It is likely to come on after severe physical strain or after the excessive use of stimulants. Among the general disorders which are regarded as causes are anemia, leukemia, emphysema, and syphilis. Hemorrhage into the vitreous humor may be the first sign of tuberculosis of the ciliary body. A form of spontaneous hemorrhage, coming on repeatedly at intervals, is sometimes seen in young persons, particularly in men who are gouty or are the subjects of vascular disturbances, or are habitually constipated.

SYMPTOMS.—Hemorrhage into the vitreous causes loss of vision, which may be only slightly reduced or completely abolished. Generally in such a case there will remain ability to count fingers or the retention of perception of light at the time of the hemorrhage. At a later period the case may show much improvement in vision or total loss of it. In some cases the patient can read print in a hesitating manner, certain parts of the field being obscured immediately after the hemorrhage. Patients often complain that objects appear red (erythropsia).

To the ophthalmoscopist the hemorrhage appears as a dark-brownish or black mass with red borders. In some cases there is so much blood in the vitreous that no fundus-reflex is obtainable. In these patients oblique focal illumination is to be employed. If a thin layer of blood is present the fundus seems covered with a bright-red veil. After absorption has progressed numerous opacities are found in the vitreous. In some cases these eyes end in glaucoma, detachment of the retina, retinitis proliferans, and iritis.

PROGNOSIS in these cases is always serious. The younger the subject and the smaller the hemorrhages, the more favorable will be the prospect for rapid absorption. Repeated hemorrhages in young persons sometimes end in nearly complete restoration of vision. In traumatic hemorrhages the prognosis will depend, not only on the amount of blood effused, but also on other concomitant lesions.

TREATMENT.—In robust persons the eyes should be bandaged. The patient should be placed in bed, and a vigorous course of purgatives with subcutaneous injections of pilocarpin instituted. The internal administration of iodid of potassium is of value. The use of eserin and repeated tapping of the anterior chamber have been recommended. In anemic individuals these severe measures must be used with caution. After recovery attention should be given to the relief of any local or general departure from the normal condition. Stimulants, excessive exertion, and late hours should be avoided. In hemorrhages occurring in young women about the time of puberty, attention should be paid to the uterine functions.

Fluidity of the Vitreous (Synchysis Corporis Vitrei) occurs as the result of chorioiditis or retinitis, and is found in high myopia by reason of the fact that in these conditions the nutrition of the vitreous is disturbed. Under these circumstances the vitreous body becomes a thin fluid of a straw color. In cataract extractions it is sometimes observed that considerable watery fluid, in excess of the amount of aqueous, flows out of the wound. In such patients the anterior part of the vitreous has become thin. With diminished consistency there may be shrinkage of the vitreous. The anterior chamber may be increased in depth from a backward movement of the lens, and detachment of the retina may be present. According to Griffith, in cases of fluidity of the vitreous the ocular tension is more often increased than diminished. Tremulousness of the iris is often present in this disease. The condition does not admit of successful treatment.

Synchysis Scintillans (Sparkling Synchysis; Synchysis Étincelant) is the term applied to a striking and beautiful appearance due to the presence of crystals in the vitreous. These usually consist of cholesterin, although margarin, tyrosin, and phosphates are sometimes found. Ophthalmoscopically the appearance is usually that of innumerable small, moving, shining particles which have been likened to a shower of gold, although cases have been observed in which the particles appeared as white, glistening, round discs, or small, cream-colored bodies. Sparkling synchysis is a senile

condition, having been observed only in persons over fifty years of age. As a rule, vision is normal. The affection may be unilateral or bilateral, and is irremediable.

Animal Parasites in the Vitreous Humor.—Three kinds of animal parasites have been found in the vitreous body. They are the cysticercus cellulosæ, which is not rare in North Germany, where raw meat is often an article of diet, but is extremely uncommon in France, Austria, England, and America; the filaria sanguinis hominis, which has been observed a few times; and the hydatid cyst, which is of rare occurrence.

Cysticercus, which is the scolex of tænia solium, gets into the eye in this way: joints of a tenia lodge in the stomach, are digested, and the eggs are set free; or the eggs get into the stomach by contaminated water or food. Embryos develop from the eggs, penetrate the walls of the stomach by their hooklets, and enter the vessels, by which they are transported to distant parts. When leaving the vessels they bore into the tissues and grow into cysticerci. In the eye the cysticercus generally develops under the retina, which it lifts up and perforates, thus getting into the vitreous. It may enter the vitreous directly through the vessels of the ciliary body (A. Kraemer). If of subretinal origin, its development can be watched with the ophthalmoscope. In the vitreous body it looks like a small bladder, usually from three to six times the size of the optic disc, and is of a bluish-white color. So long as it is alive it often moves actively. Frequently, however, the cysticercus is seen indistinctly by reason of opaque membranes, which obscure it, and under such circumstances diagnosis may be difficult or impossible. Cysticercus occurs in one eye only, but two or more may inhabit the affected organ. In many instances the patient complains only of loss of vision, or the eye is transiently reddened, and there may be photophobia. After remaining in this state for a varying period, inflammation of the anterior ocular segment will cause the patient to consult the surgeon.

Diagnosis.—If the vitreous is clear, the diagnosis of cysticercus presents no great difficulty; if purulent hyalitis is present, the diagnosis will be impossible. In making a differential diagnosis it will be necessary to take into consideration several conditions, among which are: manifest canal of Cloquet, persistent hyaloid artery, detachment of the retina, and in children neuroepithelioma of the retina.

Prognosis and Treatment.—Unless the process is checked by removal of the parasite, cysticercus sooner or later leads to complete destruction of the globe. The treatment is operative; but unfortunately the difficulties are such that removal of the parasite with retention of useful vision is possible in only few cases, while many of the attempts have ended in enucleation. Of 60 cases tabulated by Wagner, the eyeball was saved in 44, and, of these, 21 gave a good cosmetic result. In 4 cases vision equal to the counting of fingers was retained, and in 19 cases vision was improved by the operation. The method of procedure will readily suggest itself to the

ophthalmic surgeon. This will include section of the cornea, extraction of the lens, and seizure of the cysticercus with forceps; or the sclera can be opened at the infero-temporal oblique meridian, the vitreous humor freely incised, and the parasite delivered by forceps.

Formation of New Vessels in the Vitreous.—A rare condition is the development of new blood-vessels in the vitreous. They are derived from the retinal system, generally coming from the optic disc, but have been seen springing from the periphery of the retina. After running forward as fine, convoluted branches they form loops and return. Most of the cases on record occurred in syphilitic subjects, and in several of them there were

Fig. 317.—Extensive vascular growth in the vitreous humor. (HARLAN.)

("Transactions of the American Ophthalmological Society," 1889.)

hemorrhages into the vitreous. The only case of new vessel-formation in the vitreous which the author has seen occurred in a man who attempted suicide, but succeeded only in passing the bullet through both orbits. The optic nerve on one side was severed close to the globe. In this eye an intense papillitis and abundant vitreous hemorrhage was followed by the formation of a large opaque mass in the posterior pole and the subsequent development of new vessels. In some of the reported cases, under antisyphilitic remedies, the exudation has cleared and many of the vessels have disappeared. In a case recorded by Hirschberg a bluish mass of connective tissue was situated on the disc, hiding the exit of the vessels, while a vascular web, likened to the framework of a fly's wing, extended upward and inward. The treat-

ment of this rare condition consists in the internal administration of mercurials and iodids.

Pseudo-neuroepithelioma (Pseudo-glioma), a localized collection of pus in the vitreous which simulates a retinal tumor, will be considered in the following chapter.

Foreign Bodies in the Vitreous.—The vitreous frequently is the seat of foreign bodies, which pass through the cornea and lens or through the sclera. They are often small metallic substances, pieces of iron or steel, guncaps, pieces of glass, stone, shot, etc. They often cause suppurative hyalitis even if removed early; if permitted to remain they may produce sympathetic ophthalmitis. In some few cases the foreign body can be seen with the ophthalmoscope; often, however, it is invisible by reason of hemorrhage into the vitreous, and its location can then be determined by the use of the Röntgen rays. For the diagnosis and treatment of foreign bodies in the vitreous the reader should consult the chapter on the "Sclera" (page 370).

Detachment of the Vitreous, with sequent detachment of the retina, is mentioned as the result of trauma, intra-ocular tumors, old inflammatory processes in the vitreous, chorioiditis, hemorrhages, cyclitis, and posterior staphyloma. The condition is not diagnosticable until after enucleation. Some writers on ophthalmology profess to have foretold the condition in highly myopic eyes by reason of sudden diminution of vision, concentric contraction of the field, with the presence of a sharply defined, crescentic, grayish zone around the papilla, within which the retinal vessels appear bent. That detachment of the vitreous humor is a frequent condition is shown by the statistics of Jennings Milles, who found it in 43 of 345 excised eyes.

Retinitis Proliferans, which is considered in the following chapter, produces bluish-white masses adherent to the inner surface of the retina, where they often conceal the optic disc. From them numerous interlacing strands, branching in an indiscriminate manner, pass into the vitreous.

CHAPTER XV.

DISEASES OF THE RETINA.

THE retina, the intra-ocular expansion of the optic nerve, is liable to congenital anomalies, tumors, parasites, functional diseases, changes due to circulatory disturbances, inflammations, detachments, and injuries.

CONGENITAL ANOMALIES.

These include coloboma, opaque nerve-fibres, some punctate conditions of the fundus, variations in pigmentation, and peculiarities of the blood-vessels. *Coloboma of the retina* has been considered in Chapter XII with coloboma of the chorioid (page 411). A congenital anomaly which the inexperienced observer may mistake for neuroretinitis is the presence of *opaque nerve-fibres* in the retina. This condition is due to the fact that the medullary sheath of the axis-cylinders is continued into the retina and appears as bright, white, striated patches found usually above or below the optic papilla and presenting a frayed or flame-like extremity (Fig. 2, Plate IV). Exceptionally they are not contiguous to the disc, in which event they may be mistaken for fatty deposits. Sometimes the retinal vessels are partly covered by the opaque fibres. The existence of opaque nerve-fibres does not influence vision.

Punctate Conditions of the Fundus, some of which are congenital and others acquired, will all be considered later on in this chapter. *Variations in retinal pigmentation* are numerous. Isolated dots are not uncommon; groups of pigment-dots of a brown or black color, the dots being angular or rounded in shape, and occupying an area between some of the primary divisions of the vessels, are less frequently seen. They are distinguishable from pathologic dots by the fact that the remaining portion of the fundus is normal and each dot is isolated and clearly defined. Vision in such cases is normal. Larger pigment-areas of oval outline have been occasionally observed and have been likened to moles on the skin. Retinitis pigmentosa and neuroepithelioma (glioma) of the retina, which are occasionally present at birth, do not differ from the same conditions found later in life. Insufficient pigment is the condition noted in blondes and albinos. In the former the chorioidal vessels become prominent, while in the latter the observer finds a network of red vessels spread over a yellowish or whitish field. Variations in the macular region have been described in Chapter II (Plate V). *Variations in the blood-vessels* have also been mentioned in Chapter II (page 59). Extreme tortuosity is sometimes associated with

undue prominence of the optic discs, and may simulate papillitis. The vision in such patients is either normal without, or becomes normal with, the proper glasses. These patients are generally hypermetropic. The condition is discussed in the succeeding chapter under the term "Spurious Optic Neuritis."

TUMORS OF THE RETINA.

The tumors of the retina are cyst and neuroepithelioma (glioma). *Retinal cysts,* which are only rarely discovered before enucleation, are found in the detached retinas of degenerated eyes, and are due to circulatory disturbances. Liquid transuded from the retinal vessels forms cysts, which may spread for some distance in the membrane. They are of interest chiefly to the pathologist. It is only when the cysts are located far forward, and the lens remains clear, that they can be seen. They may be mistaken for tumors of the ciliary body, but the lowered tension and the history of the case will invalidate such a diagnosis.

Neuroepithelioma (Glioma) of the Retina.—This disease, formerly called glioma of the retina, fungus hæmatodes oculi, or sarcoma of the retina, in the light of modern pathology is regarded as epithelial in origin. The name neuroepithelioma was first applied to a retinal tumor by Flexner. Wintersteiner, after exhaustive microscopic study of these tumors, concluded that they arise from the neuroepithelial layer of the retina, and should be named neuroepitheliomata. According to its location and the direction of growth, the following varieties are distinguished. They exist only in the early stages:—

1. Several nodes the size of a pinhead appear in the retina; they grow only slightly toward the vitreous, but spread in the subretinal space. By confluence they form a tuberose deposit on the outer surface of the detached retina: *neuroepithelioma exophytum* or *tuberosum.*

2. The detached retina is thickened in its entire extent or in spots; the deposits remain comparatively thin and level; later by proliferation the surface becomes uneven and protuberant: *neuroepithelioma diffusum* or *planum.*

3. The new growth increases only in the direction of the vitreous; the retina remains attached to the chorioid; the mass spreads upon the inner surface of the retina, to which it is united not closely, but by processes; the vitreous surface of the growth is finely lobulated, cauliflower-like, or nodular: *neuroepithelioma endophytum.*

Pathology.—Unlike intra-ocular sarcoma, neuroepithelioma is never pigmented. The tumor grows from the two granular layers of the retina, but chiefly from the inner one. The mass is composed of small cells in a soft basement-substance. The cells consist of nuclei surrounded by protoplasm in which minute processes are often found. Some are glia-cells, others are ganglion-cells. The cells are especially numerous along the larger vessels, and this arrangement gives rise to the tubular appearance

seen in the accompanying illustration (Fig. 319). Many specimens present long cylindric cells from the neuroepithelium of the retina. These form groups inclosing a free cavity, into which the extremities of the cells project. The retina becomes irregularly thickened, folded, and detached.

Fig. 318.—Rosette of neuroepithelial cells with short protoplasmic processes. (WINTERSTEINER.)

A hyalin concretion is shown in the illustration.

Small free nodules involve both the chorioid and the vitreous humor. Degeneration of the intercellular substance occurs very early.

ETIOLOGY.—The cause of neuroepithelioma of the retina is unknown. It is a disease of childhood, no true case having been found after the sixteenth year. Cases heretofore reported of greater age have been found

Fig. 319.—Tubulose structure of neuroepithelioma of the retina. (WINTERSTEINER.)

The blood-vessels are surrounded by layers of cells; the intercellular substance is necrotic, and several calcareous concretions (stained black) appear.

to be either sarcomas of the chorioid or pseudo-neuroepitheliomata. Of 467 true cases, 314 occurred during the first three years, 62 in the fourth, and 29 in the fifth year. The disease surely is congenital in 10 per cent. of the cases, and possibly in the majority. Sex is without influence in this disease. In 25 per cent. of the cases both eyes are affected. The second

eye becomes involved independently, there being no extension of the disease via the chiasma. The disease often appears in several children of the same family. Lerche saw four cases among seven brothers and sisters and Wilson met with a family of eight, all of whom had neuroepithelioma of the retina.

Symptoms.—Usually the first symptom is a peculiar reflex from the interior of the eye, which, from its resemblance to a cat's eye shining in the dark, was named by Beer and the older authors "amaurotic cat's eye." The parents may note that the child does not see with the affected eye. In this, the first stage, there is no pain or redness, the media are clear, the pupil is somewhat dilated, and the child's health is unaffected. Ophthalmoscopic examination shows a whitish, yellowish, or reddish-yellow mass in the fundus. The growth is covered with a plexiform network of vessels and has a smooth or nodulated surface. In this stage the growth increases slowly, and months may pass before the mass fills the globe, thus completing the second stage.

Fig. 320.—Neuroepithelioma of the retina. (Wintersteiner.)

Three separate tumors are present, the smallest being at the posterior pole of the lens.

In the third stage there is increased tension. The child becomes fretful, emaciated, and cachectic. The neoplasm enmeshes all the tissues of the globe, and finally breaks out at the corneoscleral junction in front or at the optic-nerve entrance behind. Once out of the globe it grows rapidly, forming a large, ulcerated mass, which bleeds at the slightest touch. This condition was named by the older authors "fungus hæmatodes oculi." Now other organs are involved by contiguity or by metastasis. The optic nerve furnishes a road by which the growth rapidly travels brainward. Metastases may take place in the brain, cranial bones, lymphatic glands, parotid gland, spinal cord, liver, lungs, ovaries, kidneys, submaxillary gland, or spleen. The patient dies of exhaustion.

Diagnosis.—If the ophthalmoscope shows a whitish tumor, with retinal vessels coursing over it, and the tension is increased, the case probably is one of neuroepithelioma. An error in diagnosis is possible in two directions: a tumor may be present and be overlooked, or a diagnosis of neuroepithelioma may be made, the eyeball may be removed, and the micro-

scopic examination show incorrectness of the diagnosis. Hirschberg's dictum that a diagnosis between true and false retinal tumors is always possible has been found erroneous. The most careful diagnosticians have often been in error. Of twenty-four eyes removed at Moorfield's Hospital between 1888 and 1893 for "glioma," seven were "pseudo" growths. Retinal detachment and suppurative processes in the vitreous humor cause frequent mistakes. Always the history of the case is important. The parents should be questioned as to trauma, meningitis, typhoid fever, grippe, and other infectious diseases, since these are followed by diseases of the vitreous humor. If the tension is greatly increased, the case is probably one of neuroepithelioma; if the tension is decidedly reduced, it is not neuroepithelioma. Between these extremes are cases in which tension is normal or changes from time to time. The presence or absence of blood-vessels on the growth is impor-

Fig. 321.—Neuroepithelioma of the retina. (AUTHOR.)

tant; if present, the case is probably neuroepithelioma; if absent, the tumor is usually due to an exudative chorioiditis. Unfortunately, however, there are neuroepitheliomata which are not vascular; and, on the other hand, exudation into the vitreous humor sometimes becomes vascularized. The "amaurotic eye" reflex is valueless in differential diagnosis. The conditions often mistaken for neuroepithelioma, according to Wintersteiner are:—

1. Simple Detachment of the Retina.—This is comparatively rare in childhood. Although a tumor shows a more yellowish or reddish color than a detachment, which is of a bluish tint as a rule, yet it must be remembered that, with a small tumor and a large detachment of the retina, the folds of the latter can completely conceal a neoplasm; and although the newly formed vessels of a tumor generally present a course and ramifications different from those of the retinal vessels, yet, on the other hand, there are cases which show almost no vessels. Although, as a rule, a retina which

is lifted up by serous effusion vibrates and floats when the eye is moved, while a retina detached by a tumor remains at rest, yet exceptions occur. The statement that in simple retinal detachment the tension is reduced and in intra-ocular tumor it is increased must be accepted with allowances, for in the first stage of neuroepithelioma tension is normal, and, on the other hand, in serous detachment it is often increased.

2. Leucosarcoma of the Chorioid.—This is a comparatively rare disease in childhood. Of 259 sarcomas of the uvéal tract, Fuchs found 6 leucosarcomas in children under twelve years of age. In these cases the symptoms of intra-ocular tumor are added to those of retinal detachment. The diagnosis is particularly difficult if the media are opaque or if the chorioidal tumor perforates the globe posteriorly without causing retinal detachment.

3. Tubercles in the Chorioid.—Here the history of the case is important. Miliary tubercles of the chorioid are usually found near the optic-nerve entrance in the macular region. They appear as whitish-yellow masses or nodules in the stroma of the chorioid, varying in size from one-eighth the diameter of the optic disc to the size of the disc itself. By confluence they sometimes form large masses. A rare condition is solitary tubercle, which appears as a nodule and resembles a beginning neuro-epithelioma. Diagnosis is particularly difficult in cases where the vitreous chamber is filled with granulation tissue, and in consequence of secondary glaucoma scleral ectasia appears. The diagnostic difficulties are shown by two cases reported by Jung: In the first neuroepithelioma was diagnosticated and tuberculosis was found; the second was regarded as tubercular and a tumor was found.

4. Chronic Inflammatory Processes in the Chorioid and Ciliary Body.—These are the conditions most often causing error. They show retinal detachment and the presence of a fibrinous vitreous exudate, which later becomes organized. The chief points in differential diagnosis are these: *(a)* In exudative chorioiditis the color of the vitreous mass is a metallic, brass-like yellow, while in the retinal neoplasm whitish, yellowish, reddish, and green tints are seen; yet even here the metallic lustre may be observed. *(b)* Many observers state that the exudate is non-vascular, while a retinal tumor possesses vessels. Others equally competent report cases of true neoplasm in which vessels were never visible to ophthalmoscopic examination; and, on the other hand, an exudate into the vitreous often undergoes organization. *(c)* The surface of a tumor is knobbed; that of an exudate is smooth or ragged. A tumor growing into the vitreous may have a smooth surface if it presses against the lens, while an exudate may become shrunken and conglobate. In such cases it may be impossible to make a diagnosis macroscopically even after enucleation. A tumor with a smooth surface may be a neuroepithelioma growing chiefly into the retina. *(d)* Early in neuroepithelioma of the retina the tension is normal; later it is increased. In vitreous exudation it is usually diminished. Yet there are

exceptions. The author has seen one case of pseudo-glioma with increased tension. *(c)* Posterior synechiæ and other evidences of iridal inflammation are not reliable, since they may be present or absent in each condition.

5. Acute Suppurative Hyalitis producing a yellow mass behind the lens, inflammatory symptoms, and increased tension can be mistaken for a neoplasm. The rapid course of the disease, the presence of hypopyon, scleral perforation, and the discharge of pus will serve to clear the diagnosis.

6. Cysticercus in the Vitreous can scarcely be a cause of mistaken diagnosis in this country, since it is an extremely rare disease in America, although common in Germany. It appears as a bluish-white mass in the vitreous, without vessels, with normal tension and blindness.

7. Congenital Abnormalities.—In some instances eyes have been enucleated for neoplasm and examination showed persistent vascularity of the lens-capsule, and a hyaloid artery with posterior polar cataract.

Fig. 322.—Pseudo-neuroepithelioma of the retina. (AUTHOR.)

The patient was a boy, aged five years, whose right eye had presented a grayish pupillary reflex for one year. The ophthalmoscope showed the presence of a whitish mass in the vitreous chamber. No vessels could be seen on the growth or elsewhere within the eye. Vision was lost; the tension was at times normal, at other times increased. There was no redness of the conjunctiva and no pain. The diagnosis was not made positively until after the eyeball had been enucleated and sections made. The growth is a connective-tissue mass, with numerous round cells, the result of inflammatory action. There was no history of trauma in this case. The specimen shows complete detachment of the retina. The anterior chamber is much shallowed.

8. Retinitis Circinata, when occurring in children, may be mistaken for neuroepithelioma of the retina (see "Retinitis Circinata").

9. Detachment of the Retina with Dropsical Degeneration of the Visual Cells (rods and cones), according to de Schweinitz and Shumway, may exactly resemble neuroepithelioma.

PROGNOSIS.—This is always serious, but depends upon the stage of the disease. Without treatment neuroepithelioma of the retina always causes death. Early operations are followed by 13 per cent. of recoveries. If the growth has penetrated the eyeball, and particularly if perforation occurs posteriorly, death is almost sure; but operation will probably prolong life. Operation for recurrence is useless. It is said that if the disease does not return within four years the patient will be exempt.

TREATMENT.—The treatment of neuroepithelioma retinæ should be early and heroic. Any suspicious growth within the eye of a child calls for immediate enucleation and removal of the orbital part of the optic nerve as far back as possible. The eye should then be submitted to a competent pathologist for microscopic diagnosis. If found to be true neuroepithelioma retinæ, the orbital contents should be removed. This leaves a great deformity, but increases the chance of saving life. In cases where the neoplasm has already pierced the globe exenteration of the orbit should be done immediately, unless the disease has progressed so far that the patient cannot recover from the depression of the anesthetic and operation. If the neoplasm has invaded the cavities adjacent to the orbit, operation is contra-indicated. In a case with involvement of both eyes the same principles of treatment should apply.

If an eye containing a tumor still possesses some vision, it will be best not to temporize. It is wiser to sacrifice a dozen half-blind eyes containing pseudo-neuroepitheliomata than to operate on one true case too late. In all cases of intra-ocular growth the general practitioner should seek counsel of an ophthalmologist, who, in turn, should have the advice of his *confrères*.

PARASITES.

Subretinal Cysticercus.—This parasite, whose frequency depends on the geographic distribution of tænia solium, is rarely observed in Great Britain, Russia, France, and Holland. It is occasionally seen in Germany, Italy, and Hungary. Few cases of this disease have been reported in the United States. The retina and chorioid are favorite sites for cysticercus. Thus, of 29 cases of intra-ocular cysticercus in which the eye was subjected to anatomic examination, 13 were subretinal (Kraemer). When located centrally a cysticercus causes marked loss of vision. The eye usually presents normal or subnormal, rarely increased, tension. To the ophthalmoscope the larva appears as a bluish or whitish, bladder-like mass with an orange border. The location of the head is indicated by a white spot and in some cases its movement can be observed. After a time the head breaks through the retina and enters the vitreous humor, which becomes opaque. At first the vitreous opacities are transparent, movable, and of a bluish-white color. Later the whole vitreous becomes opaque, a chronic iridochorioiditis ensues, and the eye ends in atrophia bulbi. The parasite may be found at any age. The life of a cysticercus is said to be from three months to two years. The diagnosis of subretinal cysticercus is not difficult as long as the media are clear. Cysticercus occurring in a child may be mistaken for neuroepithelioma of the retina; in the adult it may simulate sarcoma of the chorioid. The prognosis is unfavorable. An early operation may result in the preservation of the globe. A few cases have occurred in which the patient after operation retained useful vision.

PLATE XV.

Diseases of the Retina.

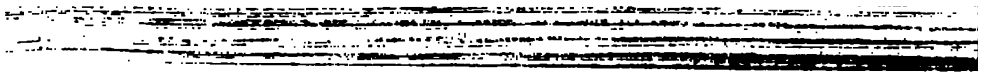
Fig. 1.—The Fundus in a Case of Pernicious Anæmia. (Oliver.)

Fig. 2.—Leucocythæmic Retinitis. (Oliver.)

PARASITES.

Subretinal Cysticercus.—This parasite, whose frequence depends on the geographic distribution of taenia solium, is rarely observed in Great Britain, France, Russia and Holland. It is occasionally seen in Germany [illegible] (Haupt). Four cases of this disease have been reported in the United States. The retina and choroid are favorite sites for cysticercus. Three of the cases of intra-ocular cysticercus in which the eye was subjected to microscopic examination, 15 were subretinal (Krusmer). When located centrally a cysticercus causes marked loss of vision. The eye usually presents normal or subnormal, rarely increased, tension. To the ophthalmoscope the larva appears as a bluish or whitish, bladder-like mass with an opaque border. The location of the head is indicated by a shiny spot, and sometimes its movement can be observed. After a time the head breaks through the retina and enters the vitreous humor, which [illegible]. At first the cysticercus capsicles are transparent, but later [illegible]

PLATE 15



Subretinal Echinococcus.—One case of this disease, that reported by Gescheidt in 1855, is recorded in ophthalmic literature.

RETINAL CHANGES FROM CIRCULATORY DISTURBANCES.

Anemia of the Retina.—Anemia of the retina is simply a local expression of general anemia, and may be exceedingly difficult of diagnosis. In fact, in the absence of a proper examination of the blood, diagnosis with the ophthalmoscope may be impossible. In some cases, however, the fundus changes are so marked as to be considered almost pathognomonic. Thus, in chlorosis, pernicious anemia, and leucocythemia the fundus often shows marked changes. The whole fundus is paler than under normal conditions; the optic-nerve head is lustreless and grayish, and neuroretinitis is common. There is a greater tendency to inflammation of the nerve-head and retina in chlorosis than in pernicious anemia, while the tendency to retinal hemorrhage is less (Stephen Mackenzie). Posey states that in initial anemia from loss of quantity of blood there are seldom ocular changes unless some other factor than loss of blood exists. In cases of chlorosis and pernicious anemia observed by Oliver there were marked changes: the optic disc was of a yellowish-white color, with hazy and in places indiscernible edges; the fibre-layer of the retina was opaque, thickened, and striated; the vessels were pallid and showed opacification of their lymph-sheaths and pronounced neuroretinitis was present (Fig. 1, Plate XV). Visual acuity was reduced and the fields of vision were contracted. Coetaneous blood-examinations showed a great diminution of red blood-corpuscles and hemoglobin.

Ischemia of the Retina.—The term ischemia of the retina indicates that the cause of the condition rests in or near the eyeball itself. Thus, ischemia follows plugging of the arteria centralis retinæ. It also has been observed in spasm of the retinal arteries (in quinin-blindness).

Ischemia of the retina may be general or partial. In embolism of the central artery, where the plug is located in the main trunk, the whole fundus is white and bloodless except such portions as are supplied by a cilioretinal vessel. An example of partial ischemia is found in case the embolus successfully traverses the central vessel and lodges in one of its branches.

The diagnosis between anemia and ischemia of the retina can be determined by taking into consideration the general condition of the patient and the ophthalmoscopic appearances (see the preceding section and "Embolism of the Central Retinal Artery").

Hyperemia of the Retina.—Retinal hyperemia, existing apart from demonstrable pathologic lesions, such as hemorrhage and exudation, is of rare occurrence, and is diagnosticated with difficulty. Hyperemia of the larger retinal vessels is indicated by increased tortuosity, which is to be expected when an elastic tube is overdistended. Unless the patient has been previously examined, the diagnosis will be in doubt, since what may

appear to be undue tortuosity may be simply a congenital condition. The calibre of the retinal vessels varies much under normal conditions. When, however, congestion of the larger vessels is accompanied by capillary hyperemia, there will be a change in the color of the whole fundus and the optic nerve will be redder than normal. Hyperemia of the retina is divisible into active and passive forms.

ACTIVE RETINAL HYPEREMIA is found in connection with inflammations of the uveal tract, and as a functional affection due to excessive use of the eyes in near work under unfavorable conditions as regards illumination, or under abnormal states of refraction or of muscle-balance. It is also found among persons exposed to excessive light and heat, as puddlers. The symptoms of active retinal hyperemia include photophobia and asthenopia. The diagnosis must rest on the results of an ophthalmoscopic examination, on the subjective symptoms, and on the history of the case. Hyperemia of the retina should not be confounded with the diffuse haziness which is due to fine vitreous opacities, or with the apparent distortion of vessels which is seen in astigmatic eyes. The treatment will consist in the removal of the cause, the proper correction of errors of refraction, and removal from unhygienic surroundings.

PASSIVE RETINAL HYPEREMIA is found wherever the egress of blood from the eyes is interfered with, as in glaucoma, brain-tumors, and other intracranial diseases producing "choked disc." Stasis hyperemia is found in mitral disease, convulsions, tetanus, and in tumors of the neck interfering with the return of blood. The symptoms are similar to those found in active hyperemia. The diagnosis is made by attention to the ophthalmoscopic findings, the results of a general physical examination, etc. The treatment will comprise the removal of the cause, when possible, and the correction of errors of refraction, etc.

Retinal Vasculitis and Perivasculitis (Sclerosis of the Retinal Vessels).—These terms are applied to inflammation of the vessel-walls or of the perivascular sheaths. The condition is shown by the appearance of white lines along the vessels. Normally the vessel-walls cannot be seen.

PATHOLOGY.—Endarteritis productiva (arteriosclerosis) begins in a thickening of the intima; if the process progresses, the condition known as endarteritis obliterans develops and the lumen of the artery is much reduced. The process is an inflammatory one, and leads to fatty degeneration and calcareous change in the intima. A granulating inflammatory process follows in the adventitia with a new growth of connective tissue. Calcification follows which may be *en plaque* or in spots. Macroscopically the first evidences of a beginning endarteritis are roughening of the intima and yellowish discoloration. Perivasculitis is a similar process which begins in the adventitia.

SYMPTOMS.—Sclerosis of the retinal vessels usually is attended by reduction in visual acuity. Hertel, however, has found arteriosclerosis in eyes possessing good vision. In such cases the findings of the ophthalmo-

scope will be of much value in the general prognosis as to the duration of life of the individual, since arteriosclerosis of the retinal vessels indicates serious lesions located elsewhere, particularly in the brain. In some cases evidences of vascular disease are presented to the ophthalmoscope when physical examination of the heart and of the arteries of the extremities fails to reveal lesions.

In sclerosis of the vessel-walls the ophthalmoscopist finds the vessels transparent, the blood-column reduced in diameter, the light-streak widened, and an interruption in the continuity of the vein at the point where it is crossed by an artery. In perivasculitis white lines appear along the course of the vessels, and in exceptional instances the vessels appear as branching white streaks.

Fig. 323.—Perivasculitis of the retinal vessels. (HARLAN.)

An area of chorioidal atrophy is seen at the macular region.

ETIOLOGY.—The vascular changes mentioned, while incident to old age, are also found in middle-aged and even in young subjects who are syphilitic, gouty, or rheumatic. In some cases trauma is a factor. In nephritis the retinal vessels are often diseased (see "Albuminuric Retinitis").

PROGNOSIS.—The prognosis in sclerosis or perivasculitis of the retinal vessels is unfavorable, there being no known means of effecting a restitution to the normal. Vision may long remain normal, or it may be suddenly reduced or abolished by rupture of a diseased vessel. The finding of changes in the intra-ocular vessels is of value in general prognosis, since the general arterial tree is often involved in the same pathologic process. In cases where the general medical examination fails to reveal cardiac, vascular, or renal disease, the ophthalmoscopic findings may be of great moment. An examination for life-insurance cannot be considered complete without an ophthalmoscopic examination.

TREATMENT.—This must be based upon general and broad lines, and includes the avoidance of excesses, the abandonment of injurious habits, the regulation of the diet, exercise, etc.

Aneurism of the Retinal Vessels.—Different forms of aneurism of the retinal vessels have been described as ophthalmic rarities. Magnus and Fuchs met with arterio-venous aneurisms produced by trauma, and the same condition has been observed as a congenital disease. Fusiform and sacculated forms have also been observed; but the commonest condition is the presence of numerous miliary aneurisms occurring in arteriosclerosis. Trauma, arteriosclerosis, and syphilis are etiologic factors. Since the retinal arteries are terminal vessels and a collateral circulation is practically impossible, it follows that rupture of retinal aneurisms is likely to occur, and probably this is the explanation of some cases of retinal hemorrhage. The diagnosis of retinal aneurism can be made with the ophthal-

Fig. 324.—Arterio-venous aneurism of the retina. (FUCHS.)

moscope. Varicosities in the veins may be mistaken for aneurisms by the inexperienced examiner. Occurring idiopathically, retinal aneurism must be regarded as significant of vascular changes in other parts of the body, particularly in the brain. The condition does not admit of treatment.

Apoplexy of the Retina (Hemorrhages in the Retina).—The differentiation between retinal apoplexy and hemorrhagic retinitis rests on the presence of signs of inflammation. A hemorrhage occurring in an eye whose retina otherwise appears normal is known as retinal apoplexy; if signs of inflammation of the retina are present, the condition is called hemorrhagic retinitis.

SYMPTOMS.—When the hemorrhages involve the macula there is great reduction in acuity of vision. Often, however, the macula is free, the hemorrhages being numerous and situated peripherally, in which case central vision will be normal. Scotomata due to such lesions will often remain unnoticed by the patient. Ophthalmoscopically apoplexy of the retina pre-

sents red or black spots, which may vary in size, shape, and number. They contrast strongly with the bright-red fundus. They may involve any of the retinal layers. When occurring in the nerve-fibre layer they present striate or flame-like shapes. Hemorrhages into the deeper retinal layers or between the retina and chorioid are rounded or irregular in shape.

ETIOLOGY.—Apoplexy of the retina, while more common among adults of advanced age, is not infrequent in both sexes about the age of puberty. The middle-aged and elderly subjects present lesions of the heart or arteries or changes in the composition of the blood. Some of the young subjects are anemic, while others show no departure from the normal. Sudden reduction of intra-ocular tension, such as follows an iridectomy made for glaucoma, sometimes causes retinal apoplexy. Some poisons—among which are serpent virus, phosphorus, and potassium chlorate—may cause the affection. Among the constitutional conditions productive of retinal apoplexy are (according to Dimmer) septicemia, pyemia, anemia, ulcerative endocarditis, leucocythemia, menorrhagia, hematemesis, hemophilia, gout, purpura, diabetes, malaria, scurvy; certain renal, hepatic, and splenic diseases; cardiac hypertrophy with valvular stenosis, atheroma of the vessels, thrombosis of the central retinal vein, and embolism of the central artery; suffocation, compression of the carotid, and hemorrhages in the newborn. Retinal apoplexy sometimes follows large cutaneous burns. It has been observed in cases of fracture of the skull.

PATHOLOGY.—Hemorrhage into the retina sometimes is due to diapedesis of blood-corpuscles; in the majority of cases, however, actual rupture of an artery or a vein occurs as a result of pre-existing perivasculitis or arteriosclerosis.

DIAGNOSIS.—This will present no difficulties to the surgeon who is familiar with the use of the ophthalmoscope.

PROGNOSIS.—While in some cases, particularly in young subjects, retinal apoplexy is followed by complete absorption of the blood and restoration of vision without scotomata, the disease in many instances is a serious affection. In many cases the apoplexy recurs from time to time. There is increase of intra-ocular tension, and the condition is then known as hemorrhagic glaucoma: a most serious disease (see Chapter XVII). In a period of weeks or months the retinal hemorrhages may undergo absorption, leaving chorioidal and retinal changes, which may be limited to small areas or may involve the entire retina. If the hemorrhage breaks into the vitreous humor, it may leave permanent opacities and lead to proliferating retinitis. Hemorrhages into the retina in elderly persons often precede cerebral hemorrhage.

TREATMENT.—Retinal apoplexy calls for attention to the patient's general health, and, when possible, removal of the cause. Stimulants should be avoided. Rest, and the use of cholagogues and purgatives will be of value. Local bloodletting from the temple is said to be helpful. Hypodermic injections of pilocarpin and the internal use of iodid of potas-

sium or bichlorid of mercury may be employed to stimulate absorption. In elderly persons a solution of eserin may be dropped into the eye twice a day.

Subhyaloid Hemorrhage (Effusion of Blood between the Retina and Vitreous Body).—The effusion of blood between the retina and vitreous humor is of rare occurrence. Hotz, of Chicago, writing in 1893, said he had observed only three cases in twenty years' practice. The disease appears suddenly and without premonitory symptoms in an eye supposed to be normal; vision is reduced to perception of light, which looks reddish or brown. The cornea and lens are found to be clear. Ophthalmoscopic examination shows the presence of a round, oval, or pyriform effusion of blood situated usually in the macular region and rarely reaching to the temporal border of the disc. Haab has seen cases in which the blood covered part of the optic disc. The lower portion of the effusion often presents a darker color. Retinal vessels in the path of the hemorrhage are hidden by it.

Etiology.—Suppression of the menses and cardiac and vascular lesions

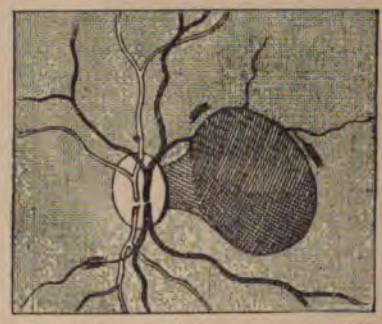


Fig. 325.—Subhyaloid hemorrhage. (Hotz.)

have been found in some of the cases, while in no small percentage not any departure from the normal could be detected.

Diagnosis.—The diagnosis of subhyaloid hemorrhage should not be difficult. A subretinal effusion of blood would not conceal the retinal vessels, which would rise over and pass in front of the effusion, and in subretinal effusion the patient would not complain of red vision.

Prognosis.—The prognosis of subhyaloid hemorrhage is favorable, vision being entirely restored with the absorption of the clot, which occurs in four or five weeks. This favorable termination may be confidently expected except in the rare cases in which the hemorrhage is of sufficient size to break into the vitreous body.

Treatment.—The general condition of the patient should be investigated and suitable remedies should be prescribed for any departure from the normal. As regards the local affection, the avoidance of stimulants and excitement must be complete. Aperients and alteratives will be in order.

Embolism of the Central Retinal Artery.—An embolus may lodge in the arteria centralis retinæ or in one of its branches.

PLATE XVI.

Diseases of the Retina.

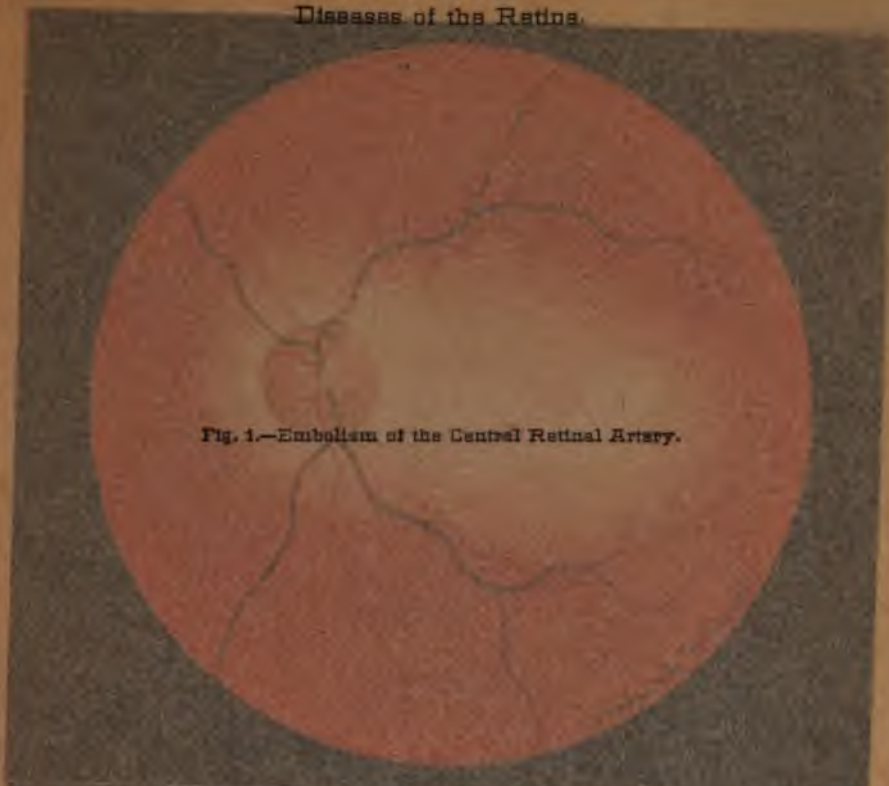


Fig. 1.—Embolism of the Central Retinal Artery.

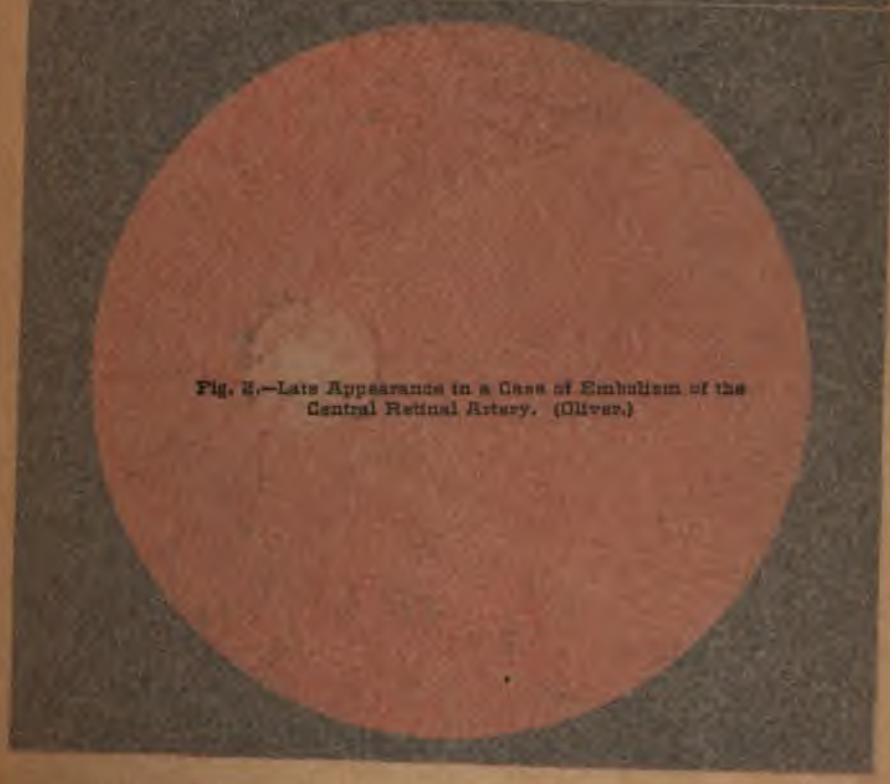
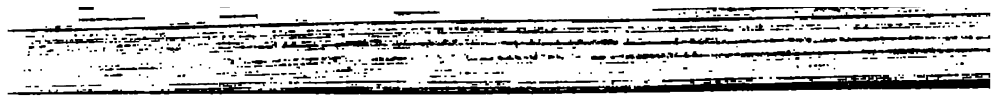


Fig. 2.—Late Appearance in a Case of Embolism of the Central Retinal Artery. (Oliver.)

PLATE 16



SYMPTOMS.—The disease comes generally without pain and without premonitory signs, although Schöbl claimed that transient obscuration of the visual field or transitory blindness are frequent premonitory phenomena. The initial symptom is sudden and complete blindness, if the central trunk is obstructed; sudden partial blindness, if the embolus passes into a branch of the central vessel. In the latter event the visual field is correspondingly limited. Complete blindness existing for several minutes or hours, if the embolus is lodged in the central artery, may be followed by a loosening of the plug, which then lodges in an arterial branch. Vision will then be restored except in the area of the visual field which corresponds to the part which the branch supplies. If examined immediately after the accident, the ophthalmoscope will show complete absence of blood from a part of or the whole retina, depending upon the location of the plug. The arteries are either completely lost to view or are much diminished in size. The optic papilla is whitish or yellowish. The veins are contracted, but may show irregular distensions toward the periphery of the retina. The veins some-

Fig. 326.—Section of the retina in embolism of the central artery. (After MARPLE.)

A, Layer of rods and cones. *B*, External granular layer. *C*, Outer reticular layer. *D*, Inner granular layer. *E*, Layer of ganglion-cells (the ganglion-cells are almost entirely absent). *F*, Nerve-fibre layer showing marked edema. The ganglion-cells have almost entirely disappeared, a few nuclei only being present. There is marked retinal edema, especially in the layer of nerve-fibres.

times present an intermittent flow of blood with or without pressure. Pressure upon the eye, by increasing intra-ocular tension, may cause a flow of blood, but there is usually complete absence of pulsation, venous or arterial, on pressure. Coagulated blood may be found in the small arteries, particularly in the macular vessels. The entire fundus is white except for the presence, in the macular region, of a cherry-red spot (Fig. 1, Plate XVI). This spot has been attributed to hemorrhage, but it is now generally believed that it is simply the natural color of the fovea made prominent by contrast with the white fundus. In a few hours the retina shows opacity due to edema. It becomes of a whitish, yellowish, gray, or greenish color except for the cherry-red spot at the macula. In rare cases of embolism the red spot may be absent; in the black races it has been observed as a black spot. Later on in the history of the case the retinal picture is that of atrophy. The arteries are extremely small, the veins are narrow and straight, the optic disc is white or of a dirty-grayish color, and there is perivasculitis and retinal degeneration. Such an eye will be blind; the pupil will be dilated and fixed and the media clear (Fig. 2, Plate XVI). In

a few of the recorded cases of complete embolism a small sector of the visual field has been preserved. In such cases cilioretinal arteries were present.

PATHOLOGY.—An embolus of the central artery lodges usually immediately behind the lamina cribrosa, at a point where the vessel is contracted and makes a turn to ascend to the papilla. Here the embolus becomes organized. In Marple's case a thrombus was found behind the embolus; the optic nerve was atrophic, the intervaginal space was dilated, and apparently there was a vaginitis. Retinal changes have been found by Elschnig and Marple, whose cases were studied in laboratories seven weeks after the onset of the disease. The inner layers of the retina—layer of nerve-fibres and ganglion-cells—are the first to become atrophic.

ETIOLOGY.—The cause of embolism of the central retinal artery is to be sought in lesions of the heart and arteries. Of 125 cases tabulated by Fischer, 70 per cent. showed valvular lesions, recent endocarditis, arteriosclerosis, syphilitic endarteritis, or aneurism. In 30 per cent. no adequate

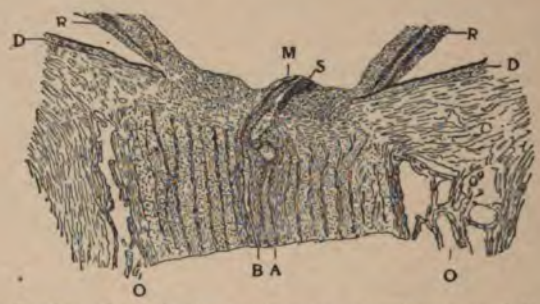


Fig. 327.—Section through the optic nerve showing an embolus in the central artery. (After MARPLE.)

A, Central artery of retina. At the bend of the vessel is seen the embolus. Behind it are a few blood-cells. *B*, Vein; *O*, *O*, Intervaginal space dilated. *D*, *D*, Chorioid. *R*, *R*, Retina. *M*, Apex of papilla. The artery here contains a large number of red blood-cells (*S*).

cause could be found, but it is safe to assume that in one-half of these cases cardiac or vascular lesions existed without giving rise to physical signs. Age is not a factor, the disease being found in the young as well as in the old. A few cases of embolism of the central retinal artery occurring in chorea have been recorded by Swanzy, Benson, Thomas, and others. Embolism also occurs in Bright's disease, and has been seen during pregnancy. Multiple emboli of septic character due to post-partum pyemia are occasionally seen. In such cases, according to Galezowski, the left eye is more often affected than the right, but both may be involved. Septic emboli produce panophthalmitis, and usually cause death. In some cases presenting the clinical signs of embolism, without discoverable cardiac lesions, it is possible that the true condition is a hemorrhage into the optic nerve-sheath. The blindness is sometimes preceded by daily attacks of epistaxis, as in cases reported by Jessup and Collins.

That embolism of the central retinal artery does not more frequently occur is doubtless due to the anatomic fact that the ophthalmic artery

branches from the internal carotid at almost a right angle, and that the arteria centralis retinæ is given off from the ophthalmic, or from one of its larger branches, in turn in much the same way. Hence, a clot leaving a cardiac valve is much more likely to continue its course in the large vessel and lodge in the brain than to enter a branch given off in this manner. If an embolus gains access to the ophthalmic artery, it is much more likely to traverse the lacrimal, supra-orbital, nasofrontal, and their terminal vessels (the ciliary arteries) than the retinal artery. It is probable that embolism of the ciliary arteries is a more common affection than has been supposed; however, owing to free anastomoses, plugging of one of these vessels is not followed by such disastrous effects as follow embolism of the central artery.

DIAGNOSIS.—The ophthalmoscopic picture described above is pathognomonic of a stoppage of the retinal circulation, but this may be due either to embolism, thrombosis, or hemorrhage into the sheath of the optic nerve. Except the embolus is lodged in a branch of the central vessel, differentiation between these conditions may be difficult or impossible. Another lesion which obscures the diagnosis is thrombosis of the central vein at a point where it can press on and occlude the adjacent artery. According to Priestley Smith, arterial thrombosis can be distinguished from embolism by a history of transient failure of sight, resembling the permanent attack in its onset. Simultaneous failure of the other eye favors the diagnosis of thrombosis.

PROGNOSIS.—This is very unfavorable. It rarely happens that the plug becomes displaced spontaneously or by treatment. If a branch of the central artery is involved, there will be loss of vision in a corresponding area of the field. Septic emboli, such as are found in puerperal fever, cause panophthalmitis and often lead to death by reason of the planting of septic foci in vital parts. It is not easy to say in these cases whether the central retinal artery alone is involved; it is likely that emboli are also present in the chorioidal vessels (ciliary arteries). In embolism of a branch of the central artery the formation of an anastomosis was observed by Königshöfer. If a cilioretinal vessel is present, the plugging of the central artery will not completely check the supply of blood to the retina: a limited area supplied by the cilioretinal vessel will functionate. While the prognosis in embolism is usually unfavorable, great or complete restoration of vision is possible. Cases have been reported in which vision was restored by deep massage combined with the internal use of Rochelle salts and iodid of potassium and hypodermic injections of pilocarpin.

TREATMENT.—The indications for treatment are (1) to dislodge and break up the embolus and (2) to promote absorption of the clot. A patient with embolism of the central artery should be sent to bed and should be subjected to an energetic course of treatment at the earliest possible moment. Hypodermic injections of pilocarpin should be given; the internal use of salines and potassium iodid should be tried; and deep massage of the eyeball

should be practiced morning and evening for a week or ten days. Hot compresses may be employed over the eye. Inhalations of nitrite of amyl may be of some use. If these measures fail, the surgeon may repeatedly tap the anterior chamber in the hope that the sudden reduction of intraocular tension will result in the dislodgment of the embolus. Massage is employed by placing the thumb or fingers on the closed upper eyelid, making deep pressure for two or three minutes. Following this the surgeon should make an ophthalmoscopic examination to determine the effect of the treatment. Sclerotomy and iridectomy have been tried in embolism with little effect.

Thrombosis of the Central Retinal Artery.—This condition may be due to cardiac or vascular lesions, or, as has been observed recently by Siegrist and Gifford, it may follow ligation of the deeper vessels of the neck. In the first of two cases observed by Siegrist after ligation of the common and internal carotid arteries the eye of the same side became blind. The ophthalmoscopic picture was similar to that which is found in embolism and in thrombosis of the central retinal artery. The autopsy showed a thrombosis of the internal carotid, ophthalmic, and central retinal arteries. In Siegrist's second case ligation of the internal and external carotids was followed by blindness, by a similar fundus picture, and by atrophy of the retina with pigment accumulation.

The ophthalmoscopic picture of thrombosis is similar to that of embolism. Premonitory symptoms in thrombosis are attacks of transient blindness in both the diseased eye and its fellow, headache, giddiness, and faintness. These symptoms are rarely present in embolism.

The diagnosis between thrombosis and embolism is difficult, not only clinically, but also microscopically. Thus, of 15 cases of supposed embolism of the central retinal artery which are recorded in literature as having been subjected to microscopic study, Marple states that in 4 no embolus was found, in 5 it was impossible to say whether the condition was embolism or thrombosis, and in only 6 was an unmistakable embolus found. It is necessary to study such specimens in both longitudinal and cross sections.

Etiology.—Von Michel attributed thrombosis to arteriosclerosis. It is possible that spasm of the muscular coat of the artery is an etiologic factor. Recently Sachs has observed the formation of a ring of constriction on one of the retinal arteries, which began on the physiologic cup and extended to the bifurcation of the artery. Similar constrictions followed at short intervals. The case was one presenting the ophthalmoscopic signs of retinal embolism and the observation was confirmed by Fuchs. Galezowski believes that, in cases of supposed embolism in which no probable source for the embolus could be found, the condition was really thrombosis due to endarteritis.

Thrombosis of the Central Retinal Vein.—A thrombus forming in the central vein or in its branches causes ophthalmoscopic appearances which were formerly credited to embolism of the central artery or to hemorrhagic

PLATE XVII.

Diseases of the Retina.

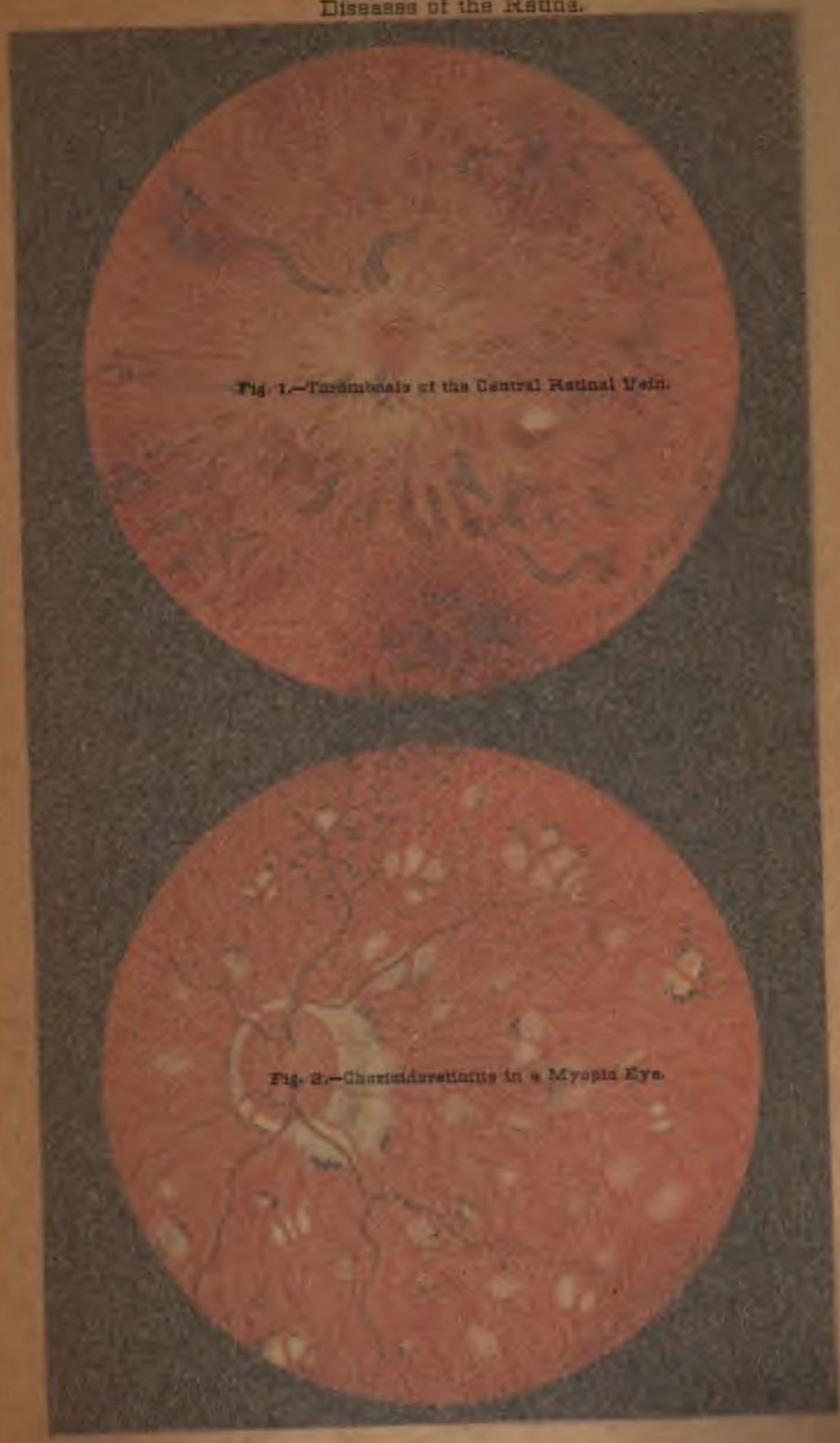
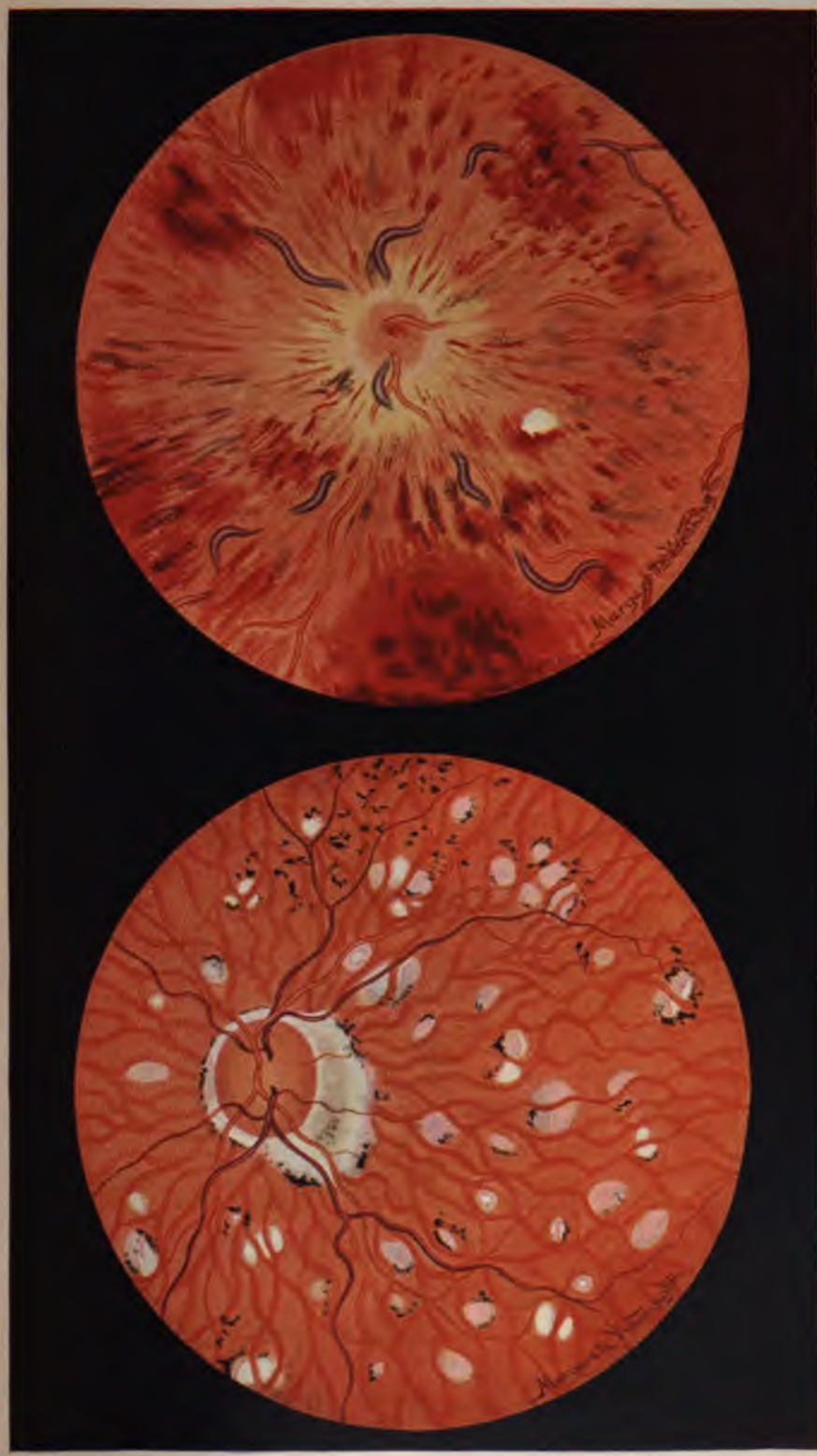
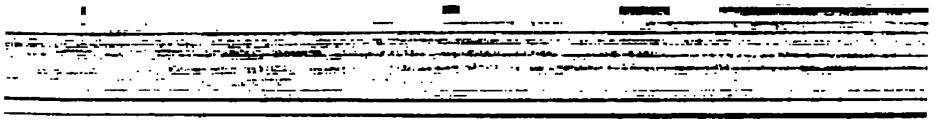


Fig. 1.—Thrombosis of the Central Retinal Vein.

Fig. 2.—Choroidoretinitis in a Myopic Eye.

PLATE 17





retinitis. Possibly some of the reported cases of hemorrhagic retinitis were examples of thrombosis. Phlebitis of the cavernous sinus, which might be expected to cause ophthalmoscopic signs, rarely does so because of the free communication existing between the orbital and facial veins.

SYMPTOMS.—In complete closure of the central vein by a thrombus the retinal veins are dilated and tortuous, the margins of the optic disc are blurred and indistinct, but the disc itself is usually not much swollen. Around the disc is a striated area of blood-extravasation into the nerve-fibre layer of the retina; and numerous hemorrhages are found in the periphery (Fig. 1, Plate XVII). The arteries are small and few in number. The veins may show on their convexity a light-streak, while the concavity is hidden in the edematous retina. Vitreous opacities soon appear, and vision becomes much reduced. In favorable cases the vitreous clears, the hemorrhages disappear by absorption, and useful vision may be retained. Some cases show in the macular region a yellowish-gray opacity presenting a cherry-red spot in its centre. Such cases simulate embolism. Vision may be completely lost in thrombosis by repeated hemorrhages. If the thrombus becomes dissolved or a canal forms alongside it, the fundus will gradually become clear.

In incomplete closure of the central vein or in closure of one of its branches, a less extensive, but similar, retinal picture is seen. There are a few hemorrhages on the disc-margin or in a peripheral part of the retina. There may be in the mildest cases only a few tuft-like hemorrhages radiating from the disc. Such cases may improve and visual acuity may be restored. Often, however, such lesions occur repeatedly, and glaucomatous symptoms supervene, the case ending in an enucleation for the relief of pain. In thrombosis of a branch of the central vein the retinal changes are limited to a corresponding area.

DIAGNOSIS.—The diagnosis of thrombosis of the central retinal vein may present many difficulties. The following table by Frost contrasts the ophthalmoscopic appearances of thrombosis and embolism:—

THROMBOSIS OF THE CENTRAL VEIN.	EMBOLISM OF THE CENTRAL ARTERY.
1. Arteries: calibre normal or slightly diminished.	1. Arteries filiform.
2. The veins are tortuous.	2. Course of veins normal.
3. Veins turgid, and appear interrupted (from being buried in the retina).	3. Veins decrease toward the disc (blood-column may be broken into segments).
4. Venous pulsation on pressure.	4. No pulsation.
5. Extensive retinal hemorrhage.	5. No hemorrhages, or very few.

PROGNOSIS.—This will depend on the location of the thrombus and whether it becomes speedily dissolved or remains, completely occluding the lumen of the vessel. In general terms it may be said that complete restoration of visual acuity is scarcely to be expected.

TREATMENT.—This is unsatisfactory. Venesection, rest, and the use of salines may be tried.

INFLAMMATION OF THE RETINA.

Inflammations of the retina may be divided into (1) simple, or serous retinitis, (2) parenchymatous retinitis, and (3) purulent (septic or embolic) retinitis. Certain affections of the retina, of which the so-called retinitis pigmentosa may be taken as the type, will be considered under the heading of "Retinal Scleroses."

SIMPLE, OR SEROUS, RETINITIS.

This affection is sometimes called edema of the retina. It is characterized by a limited or general opacity of the retina; by increased size and tortuosity of the vessels, particularly of the veins (which may be somewhat obscured by the retinal edema); and by diminution in visual acuity in that part of the field of vision which corresponds to the diseased area.

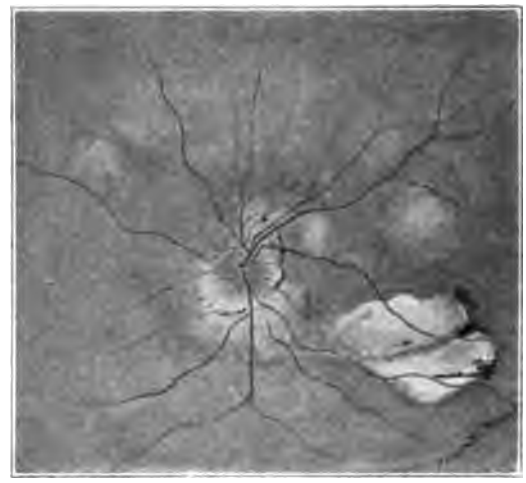


Fig. 328.—Serous retinitis. (OLIVER.)

Hemorrhages are not often found in serous retinitis. Distortion of vision may be present. Thus, objects may appear too large (megalopsia) or too small (micropsia). The patient may see better by reduced illumination or toward evening (nyctalopic retinitis of Arlt). The disease may be unilateral or bilateral. It may be caused by trauma (retinitis from concussion), by eyestrain, or by certain constitutional conditions (syphilis), or may be due to unknown causes. Other forms of retinitis may begin as serous retinitis, and will naturally be classified as such until their special features become demonstrable. The papilloretinitis accompanying sympathetic ophthalmitis is of the serous variety. Although ophthalmic writers have treated of several varieties of serous retinitis, the author will consider only one form: syphilitic retinitis.

Diagnosis.—The diagnosis of serous retinitis must rest on the ophthalmoscopic findings, the absence of appearances characteristic of the paren-

chymatous and embolic forms of retinal inflammation, and the history of the case. The blurring of the fundus found in astigmatism and the haziness due to fine vitreous opacities are sometimes causes of errors in diagnosis.

Prognosis.—The prognosis of serous retinitis is favorable.

Treatment.—The treatment will include removal of the cause, the correction of errors of refraction, the local use of atropin and smoked glasses, and attention to the general health. The correction of constipation, indigestion, etc., is of importance. A patient with serous retinitis should not be kept in a dark-room, nor, on the other hand, should he be exposed to bright light.

Syphilitic Retinitis (Specific Retinitis).—Although this disease is sometimes serous and at others parenchymatous, it will be described in this place. The question of the existence of an inflammation of the retina, caused by syphilis and independent of chorioiditis, has been much discussed by eminent ophthalmologists. In recent years the question has been decided in the affirmative. The condition known as diffuse syphilitic chorioidoretinitis, the diffuse syphilitic chorioiditis of Förster, is characterized by the presence of fine, dust-like opacities in the posterior part of the vitreous, blurring and redness of the optic papilla, and by alterations in the macular region and in the vessels.

The diffuse syphilitic retinitis of Jacobson is a later secondary manifestation of syphilitic infection, appearing six months to two years after the primary infection. The disease, however, may result from hereditary syphilis. It may be unilateral, but usually is bilateral. Other forms of syphilitic retinitis which have been described by competent observers are: (1) relapsing syphilitic central retinitis, (2) syphilitic hemorrhagic retinitis, (3) syphilitic arteritis of the retina, and (4) syphilitic perivasculitis of the retina. The first of these is a rare disease which is characterized by relapses and by ophthalmoscopic signs limited to the macula. Vision is often suddenly reduced, but improves markedly during the intervals, although after repeated attacks it may be permanently diminished. The macula looks gray or grayish-yellow, and may show small, white points arranged in groups. Pigment-spots appear after the disease has lasted for several years. Micropsia has been noticed in these cases, and sometimes the disease passes into diffuse syphilitic retinitis.

Syphilitic hemorrhagic retinitis, also a rare affection, is characterized by opacities in the posterior part of the vitreous humor and by numerous hemorrhages of various sizes and shapes. The arteries are small, and the veins are enlarged and tortuous.

In syphilitic arteritis of the retina the arteries appear as narrow gray or white bands and finally disappear, the pathologic process being an endarteritis obliterans. The veins are enlarged. The cerebral arteries in most cases are involved in a similar obliterative inflammatory process. In many of the cases few retinal symptoms are present; the vision is not much re-

duced; opacities in the vitreous, redness of the nerve-head, and blurring of its margins are not marked; and night-blindness is absent.

Syphilitic perivasculitis of the retina, also known as syphilitic periphlebitis of the retina, has been described by Scheffels. The papilla is red; the veins are enlarged and tortuous, and are surrounded by dark-red hemorrhages. The arteries may be normal and the retina transparent.

PATHOLOGY.—Nagel, who has made careful studies of eyes the seat of specific chorioidoretinitis, found localized adhesions between the chorioid and retina. About these points of adhesion the chorioid was degenerated, the choriocapillaris being entirely destroyed. Usually the retinal changes are more advanced than those of the chorioid, but the reverse may be true. The outer layers of the retina are destroyed at many points, while the inner layers show masses of irregularly arranged pigment. Müller's fibres are thickened, and the blood-vessels show marked inflammatory thickening. There are many points of similarity between the anatomic changes of specific chorioidoretinitis and those of retinitis pigmentosa.

DIAGNOSIS.—The diagnosis of these conditions must be made ophthalmoscopically and by attention to the patient's history or by the finding of other syphilitic lesions. The exclusion of other causes of retinitis will aid in the diagnosis.

PROGNOSIS.—Syphilitic retinitis is a chronic disease, although at times the onset is sudden. The prognosis will depend largely upon the time at which treatment is begun. Under energetic use of proper remedies great improvement can be expected; but when treatment has been delayed there are likely to be grave retinal and chorioidal lesions, with corresponding loss of vision.

TREATMENT.—The use of mercurial preparations by inunction or by the mouth is of the highest importance, and the remedy must be pushed to the limits of tolerance. At the same time attention should be given to the digestion and nutrition. The eyes may be protected by dark glasses, and in the acute stage a mydriatic may be used. In the later stages the use of iodid of potassium is to be advised.

PARENCHYMATOUS RETINITIS.

While in serous retinitis the changes are limited to hyperemia and edema, in the parenchymatous form hyperplasia is added to these alterations, and the deeper layers of the retina are involved. Occasionally both the serous and parenchymatous forms exist in the same eye at the same time. The causes of parenchymatous retinitis may be easily determined when the disease depends on renal or blood alterations or on cerebral lesions. In some cases no cause can be found. The pathologic changes include hyperemia, edema, round-cell infiltration, fatty degeneration, hemorrhages, and hyperplasia of connective tissue, with atrophy of the ganglion-cells. The walls of the capillaries undergo degeneration. The process may end in one of three conditions: (1) absorption, leaving the retina practically unimpaired; (2) partial atrophy; or (3) total atrophy of the retina.

The prognosis of parenchymatous retinitis is always serious. The treatment is that outlined above for serous retinitis, with the addition of such measures as will be mentioned later.

Parenchymatous retinitis may be divided clinically into the following varieties: (1) leucocythemic retinitis; (2) diabetic retinitis; (3) albuminuric retinitis; (4) gouty retinitis; (5) symmetrical macular changes in infancy; (6) circinate retinitis; (7) solar retinitis; (8) punctate conditions of the fundus.

Leucocythemic Retinitis (Leukemic or Splenic Retinitis).—Retinal changes, which occur in about 30 per cent. of cases of leucocythemia, have been divided into (1) leukemic papilloretinitis and (2) retinal hemorrhages or hemorrhagic retinitis in leukemic eyes. The disease occurs most frequently in splenic leucocythemia and as a late symptom. The characteristic features of splenic retinitis are the orange-yellow color of the fundus, the tortuosity and increased diameter of the veins, the presence of white spots with red borders in the fundus, and the presence of hemorrhages of various kinds and shapes (Fig. 2, Plate XV). Often there is papillitis, which may be slight or marked. The affection is always bilateral and is incurable. The diagnosis, if in doubt, can be confirmed by a microscopic examination of the blood.

Among the complications of leucocythemic retinitis may be mentioned chorioidal and vitreous hemorrhages, inflammation of a part or all of the uveal tract, hemorrhagic glaucoma, and exophthalmos from lymphomatous growths in the orbit.

Treatment of these cases must be directed to the improvement of the general condition of the patient, and is practically without value.

Diabetic Retinitis (Glycosuric Retinitis).—Retinal change due to diabetes is of rare occurrence and is of such doubtful individuality that some prominent ophthalmologists have questioned the propriety of assigning to it a separate place among affections of the retina. As regards the frequency of ocular affections associated with glycosuria, the reports of different observers vary greatly. Hirschberg believes that the ocular changes are often overlooked. Lagrange, among 20,000 eye cases, found 53 with diabetes; and, of 100 diabetic patients, W. O. Moore found 21 with ocular diseases, 5 having retinitis. Visual disturbances may occur in diabetes without ophthalmoscopic signs (amblyopia of diabetes). While it is usually bilateral, there are no pathognomonic signs. It is an uncommon disease, Schöbl, in a rich clinical experience, having met with only 9 such cases. From the writings of Hirschberg and others it is evident that diabetic retinal affections are divisible into five classes: (1) central punctate diabetic retinitis; (2) hemorrhagic diabetic retinitis; (3) diabetic albuminuric retinitis; (4) albuminuric retinitis in the eyes of diabetic subjects; (5) atypical diabetic retinitis. The retinal changes occur late in the disease and usually in diabetes mellitus, but have been seen also in diabetes insipidus. As a rule, diabetic retinitis shows more hemorrhages and a

smaller number of white plaques than does albuminuric retinitis. Groenouw and Uhthoff state that vitreous opacities, which are not found in albuminuric retinitis, may play an important rôle and may lead to blindness in diabetic retinitis. The descriptions given by various authors of the ophthalmoscopic appearance of diabetic retinitis are very different, and all, to a certain extent, resemble the findings in albuminuric retinitis.

In the central punctate form of diabetic retinitis the optic nerve is not affected, while in the posterior pole the fundus shows numerous ivory-white spots, streaks, or points surrounding the macula in an irregular manner. The stellate figure found in albuminuric retinitis is wanting. The spots may be round, ovoid, irregular, or semilunar in shape, and may present serrated margins. They are more frequently found on the temporal than on the nasal side of the optic disc. The white spots may remain for years. They do not coalesce, and between them are seen small hemorrhagic spots. The vitreous is clear, there is no change in the pigment, and the periphery of the fundus is unaffected. In hemorrhagic diabetic retinitis there are numerous hemorrhages, but no white spots. These cases may end in hemorrhagic glaucoma. In the third group a composite picture exists, formed of the white spots of the diabetic affection and swollen nerve-head and retina, together with the vascular changes of albuminuric retinitis. The urine of such patients presents sugar and albumin. In the fourth class the general symptoms of diabetes exist with the ophthalmoscopic picture of albuminuric retinitis. In the atypical form cases have been described which seem to be pigmentary retinitis with subsequent accidental development of diabetes. Contraction of the visual field and night-blindness are present. Schöbl saw a case in a patient whose urine showed 3 per cent. of sugar and no albumin—in which large white plaques were numerous in the periphery of the fundus, while irregular clusters of small, white spots were found in the macular region. Some cases of diabetic retinitis show atrophy of the optic nerve, hemorrhages and opacities in the vitreous, amblyopia, scotomata, chorioiditis, and hemorrhagic glaucoma.

Pathology.—In a case of diabetic retinitis Nettleship found a hyalin degeneration of the intima in the small arteries of the retina, the brain, kidneys, etc. All the layers of the retina, and particularly the nerve-fibre layer, were thickened by chronic edema and hypertrophy of the supportive connective tissue. The nerve-fibre layer showed varicosities, and the small retinal arteries presented miliary aneurisms. Von Michel has reported glycogenic degeneration of the retina in diabetes.

Diagnosis.—Frequently the diagnosis cannot be made with the ophthalmoscope alone. An examination of the urine will reveal the true nature of the case. Specific chorioidoretinitis and retinitis punctata albescens may cause confusion. Attention to the general symptoms of the patient and repeated examinations of the urine may be required to clear the diagnosis. The ophthalmoscopic differences between diabetic and albuminuric retinitis have been tabulated by Dodd, as follows:—

PLATE XVIII.

Diseases of the Retina.

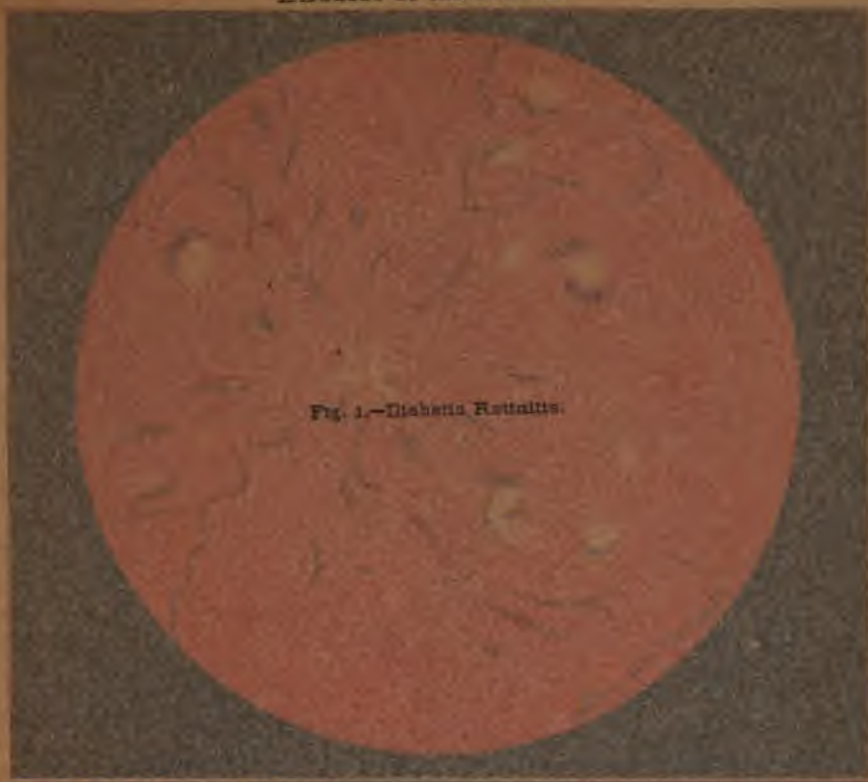


Fig. 1.—Diabetic Retinitis.

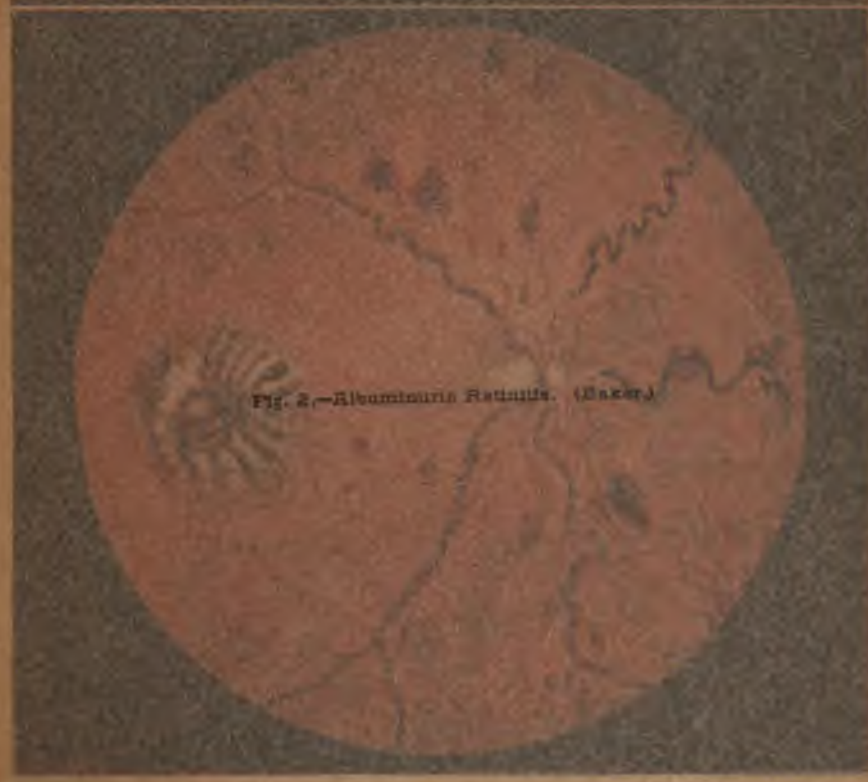


Fig. 2.—Albuminuric Retinitis. (Baker.)

Diabetic Retinitis.	Albuminuric Retinitis.
1. Groups of bright, glancing spots in the retina, irregular in outline, usually in the central part, but frequently affecting the whole of the fundus.	1. At first a group of bright, bluish-white spots is present in the centre of the retina, often forming a stellate patch about the macula.
2. If the spots are large there still exist small dots and lines, and they never run together.	2. The spots may run together and involve all of the central part of the retina.
3. The arteries and veins are not much changed in appearance.	3. The arteries are narrowed; the veins are large and irregular.
4. The optic nerve is either not affected or is atrophic.	4. The optic nerve is swollen and its outline is indistinct.
5. The retina is not diffusely affected.	5. The retina is infiltrated.

Prognosis.—As a rule, retinitis is a late and serious symptom in diabetes. While the retinal affection probably never causes complete blindness, except when glaucoma follows the hemorrhagic form, yet there is considerable loss of vision and the outlook for improvement is not favorable. While it is nearly always binocular, one eye may progress much more rapidly than the other. In the hemorrhagic glaucoma of diabetes the prognosis is most unfavorable.

Treatment.—This is simply the treatment of the general condition, and is sufficiently explained in works on the practice of medicine.

Albuminuric Retinitis (Bright's Retinitis; Renal Vascular Retinitis; Retinitis Gravidarum).—In 1827 Bright called attention to the loss of vision accompanying renal diseases; in 1856 the retinal changes were first observed in the living eye by Heymann; and in 1859 Liebreich gave an accurate description and an ophthalmoscopic picture of the fundus changes of albuminuric retinitis.

Etiology.—Of 935 cases of kidney disease, tabulated by Groenouw and Uhthoff, albuminuric retinitis was present in 209, or 22.4 per cent.; Wagner met it in 6 per cent. of his cases, while in Galezowski's cases 31 per cent. showed albuminuric retinitis. While the disease has been observed a few times in children, as a rule the patients are over 40 years of age, and the majority are between 40 and 50 years old. Men are more frequently affected than women in the proportion of about 7 to 3. As regards the form of kidney lesion present in albuminuric retinitis, the small contracted kidney is the most frequent; chronic diffuse parenchymatous nephritis (large white kidney) forms a close second; the nephritis of scarlatina is third; and the least frequent are the rare cases of amyloid degeneration. As a rule, both eyes are involved in the retinal changes, and it rarely happens that the second eye remains entirely well for the period of a year.

According to Porter, in the majority of cases of renal disease there is no disease of the retina; and a majority of renal cases showing retinal changes also show changes in the blood-vessels. He concludes that the eye disease does not depend so much on the existence of the renal affection as

on the fact that the vessels are diseased. Almost without exception the cases of albuminuric retinitis which he observed were found in syphilitics. Owing to vascular changes incident to syphilis, hemorrhages and fatty degenerations are to be expected in the retina. In nearly four thousand post-mortem examinations he has never seen one-sided nephritis. Some foreign authors have contended that in unilateral albuminuric retinitis only one kidney is diseased.

SYMPTOMS.—Persons with nephritis often complain of morning headache, nausea, vomiting, etc. The defect of vision is an early symptom, but comes with a late stage of the renal disease, and often with a dilated left cardiac ventricle. Albuminuric retinitis comes after vascular tension has been long increased and elimination begins to fail, and, although appearing as a late sign, it is often the first symptom which leads to a correct diagnosis.

The ophthalmoscopic signs of albuminuric retinitis include vascular changes, hemorrhages, white spots, exudations, and optic neuritis (Fig. 2, Plate XVIII). These changes are found chiefly in the posterior pole. The optic-nerve head is reddened, its boundaries are ill defined, the veins are broadened and tortuous, and the arteries are narrowed. The retina about the papilla looks grayish and opaque, and small hemorrhages are visible. The arteries often show white streaks on both sides of the red blood-column. In some cases papillitis occurs either in the beginning of the ocular disease or long after the appearance of the retinal changes mentioned above. In these cases probably there is often a coexistent brain-lesion with increased intracranial pressure, and a fatal result is to be expected. Papillitis, however, can occur in albuminuric retinitis without brain-lesion.

If, as is often the case, the preliminary stage of hyperemia is absent or is not observed, glittering white patches form the first sign in the retina. They are often arranged in groups surrounding the papilla like a ring and coalescing form large white spots. The inexperienced observer may mistake this condition for opaque nerve-fibres in the retina. Both present large white plaques passing from the papilla, but in the congenital condition (opaque nerve-fibres) signs of inflammation are absent. The exudation of a recent chorioiditis is to be distinguished from albuminuric retinitis by the less brilliant whiteness of the patches, and by the fact that they are accompanied by vitreous opacities and pigment collections, while hemorrhages are absent. In the macular region the white spots frequently form a stellate figure. Hemorrhages are found in great variations. In some rare instances the retinal change is present as a retinitis hemorrhagica, and the white spots are either entirely absent or are present in small numbers. In other rare cases the spots are found in the chorioid as well as in the retina, and pigmentation is present. These are cases of primary inflammation of the uveal tract. The frequency of involvement of different portions of the retina has been estimated by Schlesinger. He found hemorrhages and typical lesions in 77 per cent. of all cases of albuminuric retinitis, white spots alone in 14 per cent., and involvement of the papilla alone

in 7 per cent. The ophthalmoscopic picture is thoroughly typical when both the disseminated white spots and the stellate figure at the macula are present, but an exactly similar picture is sometimes seen, together with papillitis, in some cases of sarcoma of the brain without kidney-lesions. According to Laqueur, the same is true of some cases of diabetes mellitus, and also of patients whose urine never contained albumin. A similar picture may be found in poisoning, anemia, and syphilis. The white stellate figure at the macula is not always characteristic of kidney-lesion. On the one hand, it may be absent in albuminuric retinitis, and, on the other, it is sometimes seen in cases where albumin is never present in the urine.

The visual disturbances of this disease comprise a greater or less loss of visual acuity, without, as a rule, contraction of the field of vision and without loss of the color- and light- senses. Central scotoma is often present. The loss of vision is usually slow, rarely rapid, complete blindness being seldom observed. If it suddenly occurs in both eyes, it is suggestive of uremic amaurosis; if unilateral blindness or great loss of vision occurs rapidly, the case is likely one of embolism of the central retinal artery.

Albuminuric retinitis may show improvement or even complete cure, but many patients die before the retinal disease undergoes retrogressive changes. If the kidney-lesion improves, the surgeon may hope for improvement in the ocular condition. In some cases the retina improves regardless of the extension of the renal affection. Retinal improvement, when it occurs, is slow, and the stellate appearance of the macula is the last to disappear. The optic-nerve head may become whitish and atrophic and useful vision may remain. Some cases show alternating loss and improvement of vision for a long period. Complete blindness as a result of this disease is extremely rare. Complications are not common, and include detachment of the retina, hemorrhage into the vitreous humor, hemorrhagic glaucoma, and embolism of the central retinal artery.

PATHOLOGY.—The anatomic changes in albuminuric retinitis are found chiefly in the posterior pole, rarely reaching forward as far as the equator, and never involving the ora serrata. They consist of edema and inflammatory deposits. The edema accounts for the diffuse cloudiness which is visible ophthalmoscopically. The blood-vessels, not only of the retina, but also of the uveal tract, frequently show sclerotic or hyalin changes in their walls. The radial fibres of Müller undergo proliferation, and their interstices are filled with a fibrinous deposit. In the retinitis albuminurica of pregnancy changes in the retinal vessels are occasionally absent (Silex).

Hemorrhages occur into the retina and at times break through into the vitreous humor. The white spots, which are visible with the ophthalmoscope, depend on granular or fatty degeneration of the supporting tissue (Duke Carl Theodor, Leber); upon fatty degeneration of cells, particularly in the two granular layers; or upon the presence of foci of varicose hypertrophied nerve-fibres. The stellate figure in the macular region de-

pends chiefly upon fatty degeneration of Müller's supporting fibres, which radiate from the fovea centralis, and here do not, as in other places, push through the retina. Weeks questions this explanation. His researches lead him to believe that the changes in Müller's fibres are due to post-mortem alterations. The chorioid on microscopic examination shows similar changes, viz.: lesions of the vessels and inflammatory changes.

In the optic nerve, together with hypertrophy of the connective tissue, there is edema or pronounced inflammation. Generally the inflammatory changes involve only the papilla and end at the lamina cribrosa, without further extension centrally. Gurwitsch found hyalin bodies in the head of the optic nerve and in the granular layer of the retina.

The cause of retinal detachment in albuminuric retinitis is explained by Leber and Nordensen in this manner: the small elevations, especially around the papilla, depend on a primary exudation into the subretinal space, while further detachment can be attributed to shrinking of the vitreous humor.

DIAGNOSIS.—In typical cases the diagnosis of albuminuric retinitis is not difficult, but it must be remembered that, while the ophthalmoscopic picture is highly suggestive, it is not pathognomonic, similar fundus changes being met with in rare instances in cases of intracranial tumor, lead encephalopathy, pachymeningitis hemorrhagica, anemia, syphilis, and diabetes mellitus.

Tumor of the Brain, as a rule, is easily distinguishable from Bright's disease. However, in some cases of cirrhotic kidney there is little or no dropsy, headache and vomiting are often prominent and distressing symptoms, and pain may be most severe in the back of the head; epileptiform convulsions and even hemiplegia may be present, while the ophthalmoscopic changes may be similar to those found in cerebral tumor.

In the differential diagnosis it will be necessary to give attention to the following points:—

1. The condition of the urine. While the finding of albumin and tube-casts will clear the diagnosis, in some cases of cirrhotic nephritis albumin may be scanty or entirely absent and tube-casts may be so few as to escape detection.

2. Condition of the heart and great vessels. In all forms of Bright's disease, except possibly in the pure form of waxy kidney, the blood-pressure is high, the arteries are atheromatous, and the left ventricle is hypertrophied. In the absence of a valvular lesion these symptoms are highly suggestive of renal disease.

3. Ophthalmoscopic changes. "The ophthalmoscopic changes, *per se,* can never be taken as an absolutely certain guide, for the appearances typical of one condition are, in some rare and exceptional cases, met with in the other." (Bramwell.)

Saturnine Retinitis.—While in most cases of chronic lead poisoning (which may present ophthalmoscopic changes similar to those of albu-

minuric retinitis) albuminuria is present, exceptional cases have been observed by Förster, Rosenstein, and Lehmann. The diagnosis between albuminuric and saturnine retinitis must rest on the following data: (1) the history of the case with special reference to the patient's occupation; (2) the presence of a blue line on the gums in case of lead poisoning; (3) the finding of lead in the urine.

Anemia and Syphilis.—In cases of these diseases presenting ophthalmoscopic appearances similar to those found in albuminuric retinitis, the diagnosis may be established by an examination of the blood in the one and by the history and lesions of the other.

The ophthalmoscope cannot always make a distinction between albuminuric retinitis and the retinitis associated with diabetes. Here an examination of the urine will give correct information. Cases of hysteric blindness occurring during the puerperium, especially if a trace of albumin is present, may cause difficulty in diagnosis. Here, however, there will be an absence of ophthalmoscopic changes. In all cases of suspected albuminuric retinitis repeated examinations of the urine should be made for albumin and casts. In case a mydriatic is used to facilitate the ophthalmoscopic examination, the surgeon should use euphthalmin or homatropin, since their effect lasts only a short time. If atropin is used and the patient suffers further diminution of vision from progression of the disease, the surgeon may be unjustly blamed for the loss of sight.

PROGNOSIS.—Albuminuric retinitis is an indication of a serious underlying condition, and its appearance usually indicates the early death of the patient. The prognosis is most unfavorable in chronic nephritis; it is more favorable in acute nephritis and relatively most favorable in the albuminuria of pregnancy. Probably 85 per cent. of all persons with albuminuric retinitis die within two years. A few live for three, four, five, or six years, and exceptional cases have survived for ten or twelve years. The social *status* and hygienic surroundings have an influence on the duration of these cases, the poor dying sooner than the wealthy.

The Retinitis Albuminurica of Pregnancy presents many peculiarities, on which account it must be considered alone. Comprehensive descriptions of this disease have been given by Axenfeld and Silex. The latter observed thirty-five cases in seven years. In this disease the loss of vision appears slowly in the course of weeks and months, and chiefly in primiparæ and in the latter half of pregnancy. Exceptionally the retinitis may appear first in childbed. Visual acuity diminishes gradually in both eyes without contraction of the visual field, and without loss of the color-sense. Sometimes complete amaurosis occurs, particularly if eclamptic seizures accompany the disease. This blindness is traceable to uremia, and usually disappears. It is only when complications occur, such as detachment of the retina, that vision can be completely lost. In subsequent pregnancies mild recurrences are the rule, by each of which a further loss results. Disturbance of the general health may be absent; only edema is present.

The ophthalmoscopic signs are the same as those found in albuminuric retinitis from other causes. As an early symptom Silex mentions a change in the central reflex streak of the arteries, which, in the upright image, appears broadened and of a glistening golden yellow. Yet this phenomenon, as Silex especially states, is found also in syphilis and in arteriosclerosis, and hence is not characteristic of the albuminuric retinitis of pregnancy. The changes in the vascular reflex are attributed to an engorgement in the lymph-sheaths of the blood-vessels.

Retinitis gravidarum appears, according to Groenouw:—

1. In the pregnancy kidney, in which there is fatty degeneration of the epithelium of the uriniferous tubules. The urine in these cases is passed in diminished quantity and is of dark color. It is strongly albuminous, showing hyalin, rarely granular, casts, with fatty degeneration of the epithelial cells. The disease occurs in from 1 to 20 per cent. Retinitis albuminurica, according to Silex, occurs about once in three thousand pregnancies. Thompson found, among thirty cases of retinitis albuminurica observed by himself, four that were due to pregnancy.

Furthermore, albuminuric retinitis can occur:—

2. If an acute nephritis accidentally originates during pregnancy, or, finally:—

3. If an old chronic nephritis becomes unfavorably influenced by pregnancy. In the two examples last named, pregnancy is not the cause, but is only a complication, of the kidney affection. The pathologico-anatomic findings in the albuminuric retinitis of pregnancy are the same as those found in other instances, only involvement of the vessels is either entirely absent or is insignificant.

The prognosis as regards life and vision in cases of albuminuria is most grave when a chronic nephritis exists, since most of these patients die within two years; it is better in acute nephritis, since complete cure of the eye and kidney symptoms may occur; and is most favorable in pregnancy kidney (albuminuria due to pressure). Of twenty-one cases of retinitis gravidarum, Silex saw a complete disappearance of the retinal changes in two cases; the other cases presented permanent retinal changes either in the form of small white spots or as pigment degeneration in the macular region. Persistent loss of vision points to a complication, such as retinal detachment or atrophy of the optic nerve. Partial atrophy of the optic nerve was observed by Förster in three of four women with retinitis gravidarum who were delivered during eclampsia.

If, however, premature labor is induced, the prognosis is much more favorable as regards vision. Without the induction of premature labor the prognosis is most serious. Howe, in an analysis of cases extending over a period of fifteen years, says: "These tend to show that, when the vision begins to be impaired only in the last two weeks of pregnancy, recovery follows almost invariably. Of those described as being in the eighth month or thereabouts, when the retinitis commences, not one-half recovered, and

several did not materially improve. Again, when this began earlier than was estimated,—as the middle of the seventh month, when Nature did not interfere by bringing on a miscarriage, and when the patient escaped with her life,—it was only to remain blind forever afterward."

TREATMENT.—The treatment of retinitis albuminurica must be governed by the fundamental disease. Sudorific and purgative treatment will be in order. A long-continued use of potassium iodid, citric acid, and similar remedies has proved of value. The diet should be nutritious but non-stimulating, milk diet being preferred. The patient's strength should be kept up by preparations of iron. If the cause of the kidney-lesion can be determined,—such as malaria, syphilis, or lead poisoning,—it should receive appropriate treatment. The use of mercurials, however, must be carefully watched lest harm result. Cupping may be used, but is of doubtful value. The use of alcohol, coffee, tea, and tobacco is to be interdicted. To improve the retinal circulation efforts to reduce intra-ocular tension by means of instillations of 1-per-cent. strength solution of pilocarpin or $^1/_2$-per-cent. strength solution of eserin may be tried.

The albuminuria of pregnancy demands careful attention from the family physician. The question whether premature labor should be induced should receive careful consideration. Many surgeons believe that where there is progressive failure of vision from retinal changes premature labor is justifiable; and, when a preceding pregnancy has left the patient with permanent loss of vision, abortion should be produced.

Gouty Retinitis.—Elderly subjects with the gouty diathesis often present lesions of the retina and retinal vessels. There is usually progressive impairment of vision of both eyes. Bull states that while the loss of central vision is marked, peripheral vision is little impaired, and gouty retinitis never ends in blindness. Ophthalmoscopic examination shows patches of yellowish-white exudation in the macular region and adjacent to the optic disc; hemorrhages, which occur for the most part in the early stages of the disease; opacity and thickening of the vessel-walls; and irregularity in the calibre of the arteries and veins. These patients show general arterial sclerosis. The urine is of high specific gravity. It contains an excess of uric acid and some albumin, but no casts are found in uncomplicated cases. The ophthalmoscopic picture is not sufficiently characteristic for diagnosis, which, however, can be readily determined by exclusion. The pathologic changes in this disease include extensive arteriosclerosis and phlebosclerosis, with obliteration of the smaller arterial branches; degeneration of the retinal layers and particularly thickening of the nerve-fibre layer of the retina from the presence of granular bodies; varicosities of nerve-fibres and changes in the chorioidal vessels similar to those found in the retina. The prognosis is usually favorable as regards the retention of the amount of vision existing at the time of examination, but much depends upon the willingness of the patient to adopt dietetic and medicinal treatment. Many of these patients die from cerebral hemor-

rhage. The ocular treatment will include rest and the wearing of proper glasses. The general treatment is sufficiently described in works on the practice of medicine.

Symmetrical Macular Changes in Infancy (Infantile Amaurotic Family Idiocy; "Rare Fatal Disease of Infancy with Symmetrical Changes in the Macula Lutea," Kingdon).—In 1881 Waren Tay reported the case of a child, aged 12 months, who was mentally deficient and who presented symmetrical changes in the macula. When first examined the optic discs were normal, while at the macula there was a diffuse white plaque, in the centre of which a brownish-red spot stood out, thus resembling the ophthalmoscopic picture of embolism of the central retinal artery. Five months later the spots remained the same, but the optic discs were atrophic. Since Tay's observation about seventy cases have been reported. The chil-

Fig. 329.—Macular change in infancy. (Tay.)

dren are born healthy; at the end of several months sluggishness and somnolence are noticed; there is gradual loss of vision; the child makes irregular and purposeless movements; it is unable to hold itself erect; the indications of weakness increase, and the end is death by marasmus. Only one case is known to have lived longer than two years. The ophthalmoscopic picture as described above is always present, and in addition thereto some cases show nystagmus, strabismus, or oculomotor paralysis. The etiology of the disease is unknown, but most of the recorded cases have occurred in Jews or Poles. Pathologic studies by Sachs, Hirsch, and others have not determined the nature of the disease. Sachs, in his first case, found the cerebral fissures uncommonly pronounced, and the cerebrum resembled that found in a lower order of development. While the chiasma, pons, and medulla and great ganglia were normal, the great pyramidal cells were lacking or were markedly degenerated in the cortex. Changes

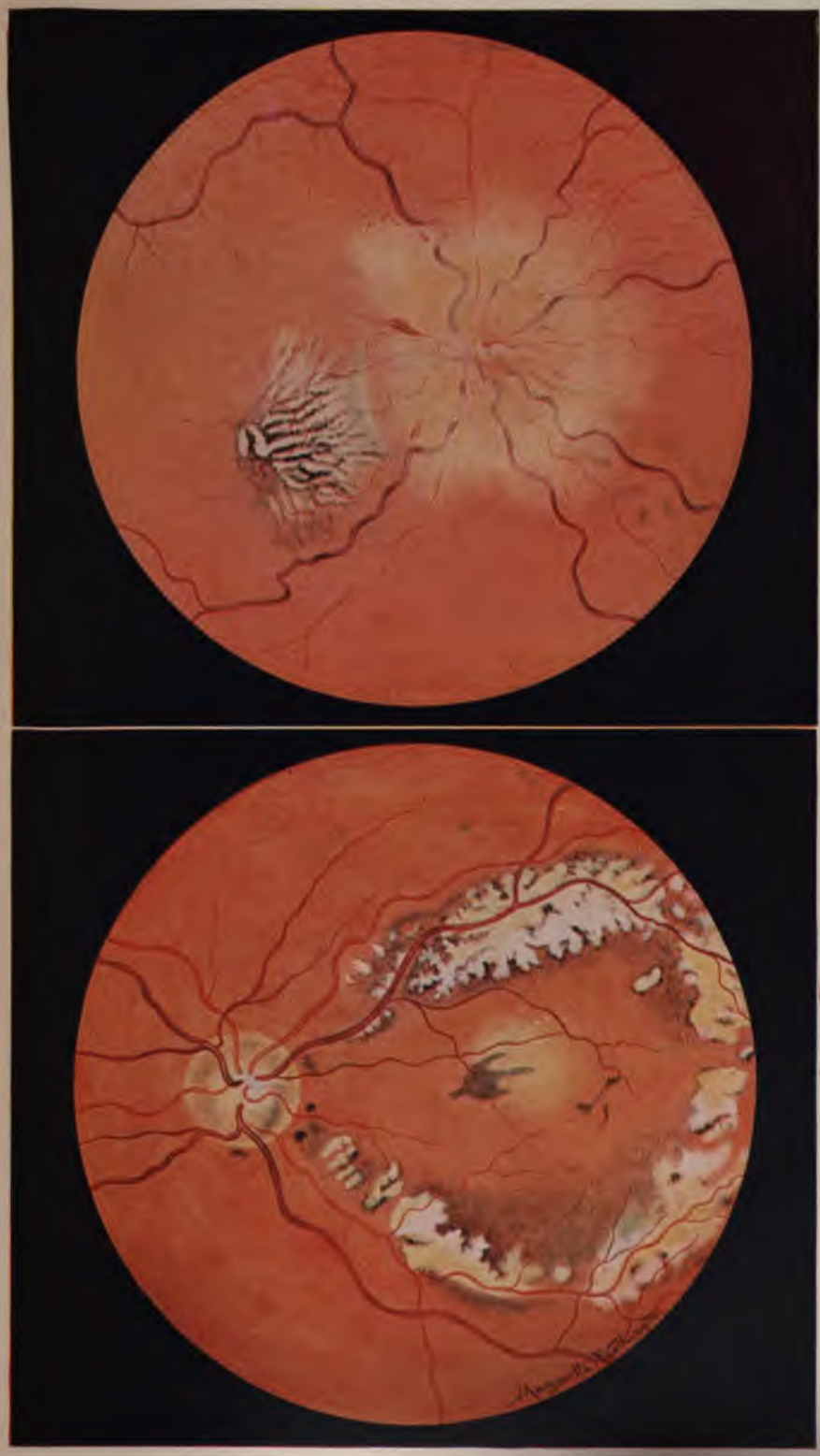
PLATE XIX.

Diseases of the Retina.

Fig. 1.—Albuminuric Neuroretinitis in a Child with Congenital Syphilis. The lesions in the macular region were attributed to syphilis. (Leopold.)

Fig. 2.—Retinitis Circinata. (De Schweinitz.)

PLATE 19



were also found in the spinal cord and retina. Holden found degeneration of the ganglion-cells of the retina. Sachs has recently examined another case. This showed some deficiency in the development in the cerebral white fibres and degeneration of the pyramidal tracts in the spinal cord. While there was an increase in the neuroglia-cells, the most marked change was found in the large ganglion-cells of the entire nervous axis. The cells showed disintegration, shifting, or entire absence of the nucleus, and the formation of pericellular spaces. Sachs regards the changes as due to an arrest of development, while Kingdon and Russell look on them as due to a degenerative process. Hirsch regards the disease as an acquired affection involving the nerve-cells of the entire system, produced by some kind of toxic agent. Treatment of this disease must be wholly symptomatic.

Retinitis Circinata (Circinate Retinitis).—This rare form of retinitis was first accurately described by Fuchs in 1893, who observed it in 12 of 70,000 patients. It is characterized by the appearance, in the macular region, of a circular or oval zone of yellowish-white exudation, which, with indented edges and pigmented borders, often resembles a wreath (Fig. 2, Plate XIX). Often the macula itself is diseased, showing irregular pigment patches resting on areas of yellow exudate. The retinal vessels pass over the circinate deposits. The remainder of the fundus is normal. Vision is gradually reduced in these cases, and the visual field is slowly limited. A central scotoma is present; the light-sense is unaltered; metamorphopsia, night-blindness, and day-blindness are absent. The disease is eminently chronic, and the ophthalmoscopic picture may remain unchanged for years. On the other hand, de Wecker has observed new hemorrhages with the formation of new white spots. In Fridenberg's case new vessels formed in the retina. De Wecker saw circinate retinitis in one eye and hemorrhagic retinitis in the other of an aged diabetic subject. Vitreous opacities of various sizes, retinal detachment, and thickening of the retina at the macular region have been observed in these cases.

Etiology and Pathology.—The etiology of circinate retinitis is unknown. The disease seems, however, to depend on arterial sclerosis. Syphilis may be an etiologic factor. The age of the accurately recorded cases has varied from 6 to 77 years. The disease has been seen more often in women than in men. In 5 of the 12 cases observed by Fuchs the changes were symmetrical; in 7 only one eye was affected.

Fuchs regarded the white patches as fibrinous exudates into the deeper parts of the retina. De Wecker attributed the changes to fatty degeneration, the result of hemorrhages. Ammann, after having made the anatomic examination of an eye which at one time presented a typical picture of retinitis circinata, concluded that the white spots are brought about by fat-cell clusters sequent to hemorrhages.

Diagnosis.—The ophthalmoscopic picture is so typical that the diagnosis should not present much difficulty. However, de Wecker has called attention to the danger of mistaking this disease, when it occurs in chil-

dren, for neuroepithelioma of the retina. He cites the case of a girl, 6 years of age, in whom a *confrère* removed an eye which for two years had presented lesions resembling neuroepithelioma of the retina. Examination showed the presence of plaques of atrophied chorioid and degenerated retina, as well as a small ossifying fibroma.

PROGNOSIS.—Although the disease is incurable, in none of the recorded cases has the patient become entirely blind.

TREATMENT.—This must be carried out on general principles, tonics or antisyphilitic remedies being used as indicated. Errors of refraction should be corrected.

Retinitis from Exposure to Excessive Light.—Conjunctivitis, keratitis, and retinitis may be produced by exposure to excessive light, either from looking at the sun (solar retinitis), from reflection from the snow (snow-blindness), from the injurious effect of electric light (electric ophthalmia), or from a flash of lightning.

SOLAR RETINITIS.—Blinding of the retina from looking at the sun has occurred in a number of cases, chiefly from the observation of solar eclipses. Following the exposure the patient will complain of a central scotoma, which may be absolute. Central vision for colors is also defective. Distortion of objects is also a symptom. Visual acuity may be slightly or greatly reduced. The duration and extent of these symptoms will depend upon the length of exposure and the intensity of the sunlight. Ophthalmoscopic changes may be absent, or may include loss of the macular reflex, the presence of a small spot of an orange color near the fovea, with alterations in pigmentation. The prognosis in solar retinitis must be guarded. Severe cases do not improve. Swanzy states that hitherto no case in which vision was reduced to less than one-third has recovered full visual acuity. The treatment will include rest, the wearing of dark glasses, the hypodermic injection of strychnia, and the use of the constant galvanic current.

SNOW-BLINDNESS.—Exposure of the eyes to the reflection from snow causes ocular changes, and may lead to permanent blindness. Usually, however, the lesions are limited to the conjunctiva and cornea. There is intense photophobia, blepharospasm, and sometimes chemosis, together with the ordinary evidences of conjunctivitis. The cornea may present dendriform ulceration. Some cases complain of the sensation of a foreign body in the conjunctiva. Noyes states that the retina may be anesthetic or hyperesthetic. The prognosis is favorable. The treatment includes rest, the wearing of smoked glasses, the use of holocain drops, and cold applications.

ELECTRIC OPHTHALMIA.—Exposure to flashes of electric light, during electric welding or from the short-circuiting of the current, may produce conjunctival, corneal, and retinal changes. The same changes may be found in electricians who use a strong arc light. In a few minutes, or perhaps several hours after exposure, the patient will complain of burning

pain in the eyes, photophobia, blepharospasm, swelling of the lids, and perhaps reduction in vision. The pupil is strongly contracted. These symptoms are followed by a muco-purulent conjunctival discharge. Examination may show contraction of the field of vision, the presence of a small scotoma, congestion of the retinal veins, and slight haziness of the retina. Rivers saw a case in which there was exfoliation of the corneal epithelium and retinal opacity with great reduction in visual acuity. The patient's face and eyebrows were burned.

While the prognosis is usually favorable in these cases, in the severer types of injury there may be permanent reduction in visual acuity, and pain and photophobia may be of long duration. Usually rest, the wearing of dark glasses, and the use of atropin will be followed by an early recovery. Pain in the acute stage may be relieved by the instillation of holocain and the application of cold compresses.

Punctate Conditions of the Fundus.—Occasionally, in eyes apparently normal, minute white or yellow dots are seen, either single or arranged in groups. The width of one of these dots usually does not exceed that of the retinal vessels. Some of the punctate conditions of the fundus doubtless take their origin from the chorioid, while others begin in the retina. Although these punctate conditions cannot be properly classified, some authors attributing them to the chorioid and others to the retina, they will all be considered in this place. According to Frost, they are chiefly: (1) Gunn's dots; (2) metallic dots; (3) isolated dots; (4) retinitis punctata albescens; (5) Tay's chorioiditis; (6) colloid change in the macular region (this has been described in Chapter XII); (7) diffuse dots resembling Tay's chorioiditis.

1. Gunn's Dots ("Crick's" Dots).—These are small whitish or yellowish dots which may be single, but generally are found in clusters in the region of the yellow spot. They are seen with difficulty, and are found chiefly in young subjects. They are without pathologic significance.

2. Metallic Dots.—These occur singly, and may be present in any part of the fundus. They are brilliant, and have been likened to particles of mercury. Frost has found them chiefly in cases where the ophthalmoscope was used to find the cause of an unexplained amblyopia, but they are also seen in normal eyes. They are supposed to be due to an irregularity of the retinal surface, producing total reflection, the metallic appearance being caused by the image of the mirror of the ophthalmoscope.

3. Isolated, or Neuritic, Dots.—These are found in the foveal region in cases of neuroretinitis, and are as white as chalk and of irregular shape.

4. Retinitis Punctata Albescens.—Under this name Mooren described the case of a man, 30 years of age, whose fundus presented hundreds of white spots distributed equally. The spots are small and of white or yellow color, without pigment borders. They never coalesce, and are rarely found in the foveal region. The disease causes reduction in central vision,

while peripheral vision is normal. Night-blindness is absent. The few cases which have been studied occurred in young subjects, and the disease has been found in several members of the same family. It is supposed to be either a congenital affection or one that develops in early infancy.

Nettleship has described a somewhat similar affection in which minute, white, round spots were scattered over the fundus and were associated with pigment changes at the periphery and with night-blindness. They are known as Nettleship's dots. They are stationary or slowly progressive. Some authors have regarded this condition as a variety of retinitis pigmentosa, but this view is generally considered erroneous.

5. Tay's Chorioiditis, also known as central senile guttate chorioiditis, or punctate retinitis, includes a group of cases characterized by the presence of round, light-colored dots, which are found chiefly in the central part of the fundus. The dots are small, their diameter equaling that of the primary retinal arteries. They occur chiefly in middle-aged persons. When the dots are numerous, this disease causes some loss of vision.

6. Colloid Change in the Macular Region.—This condition is described in Chapter XII, and is regarded as belonging to chorioidal diseases.

7. Diffuse Dots resembling Tay's Chorioiditis.—In this condition numerous dots, which are not limited to the macular region, are found in the eyes of young subjects, and remain for many years without appreciable change. In some of the cases, however, night-blindness and some contraction of the visual field have been observed.

Prognosis and Treatment.—The punctate conditions of the fundus do not admit of removal. They do not often lead to much loss of vision. These cases should receive general attention. Errors of refraction should be corrected, if present. It may be advisable to limit the amount of near work.

PURULENT RETINITIS.

Until recently the possibility of the occurrence of purulent retinitis apart from purulent chorioiditis has been denied. Clinically cases of purulent retinitis may be divided into four classes: (1) purulent traumatic retinitis, (2) purulent secondary (or induced) retinitis, (3) purulent metastatic (or embolic) retinitis, and (4) septic retinitis of Roth.

Purulent Traumatic Retinitis results from infection following perforating wounds or the lodgment of foreign bodies. The clinical picture is essentially that of suppurative chorioiditis (panophthalmitis).

Purulent Secondary Retinitis follows upon perforating ulcers of the cornea. The clinical picture is that of panophthalmitis.

Purulent Metastatic, or Embolic, Retinitis may arise in the course of any one of many infectious diseases. The symptoms may be acute, resembling those of panophthalmitis, or chronic, resembling pseudo-neuroepithelioma of the retina. The anatomic changes in these affections may

PLATE XX.

Diseases of the Retina.

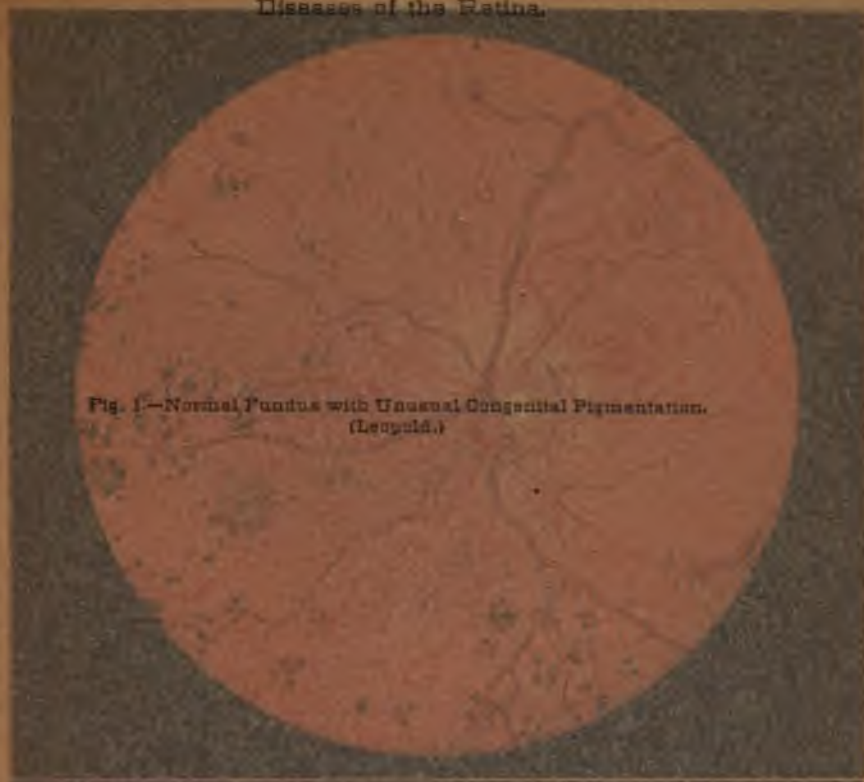


Fig. 1.—Normal Fundus with Unusual Congenital Pigmentation. (Leopold.)

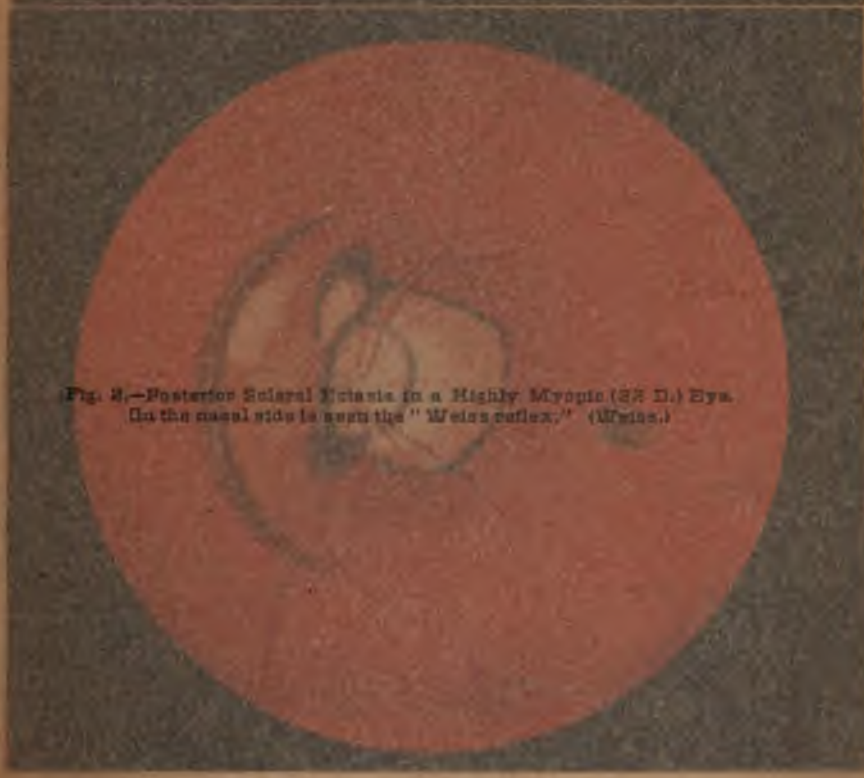


Fig. 2.—Posterior Scleral Ectasia in a Highly Myopic (22 D.) Eye. On the nasal side is seen the "Weiss reflex." (Weiss.)

PUBULENT RETINITIS.

PLATE 20

begin as a purulent retinitis, a purulent chorioiditis, or as a chorioido-retinitis. A form of metastatic retinitis which was first described by Roth as septic retinitis will now be considered.

Septic Retinitis.—To external examination an eye with septic retinitis shows nothing abnormal except possibly conjunctival hemorrhages. The dioptric media are clear. Characteristic changes found in the retina, in the neighborhood of the papilla and macula, are hemorrhages and white spots (Roth's spots), which vary much in shape and number. There may be a single spot or the foci may be numerous. The white spots may exist entirely apart from the hemorrhages or may lie within or adjacent to the latter. They never assume the stellate figure so often found in the macular region in cases of albuminuric retinitis; and, as a rule, they do not tend to become larger. They are absent in one-third of the cases, and rarely appear alone without hemorrhages. The hemorrhages are found chiefly along the large vessels, and seem to be of venous origin. Large preretinal effusions of blood are frequently found. Marked signs of inflammation are absent, the papilla being generally well defined. The retinal vessels are usually not changed in calibre, although the veins may be tortuous. In exceptional cases only one eye is involved. One eye may present the picture of septic retinitis while the other shows the changes of purulent chorioiditis. As a rule, there is no great reduction in visual acuity. In some cases, however, the general weakness of the patient is so great as to prohibit an accurate examination of vision. Often the advent of the retinal changes cannot be determined with certainty. Gimurto has observed the disease as early as the fifth and as late as the twenty-third day after confinement. Septic retinitis has been observed in from 33 to 87 per cent. of cases of sepsis. It is found in all forms of sepsis, but is particularly frequent in septicemia. The disease runs a slow course, and, if the patient's life is spared, complete recovery of the eye may occur. Although at the beginning the signs of metastatic chorioiditis are similar to those of septic retinitis, a differentiation can soon be made. Metastatic chorioiditis runs a rapid and destructive course with marked inflammatory symptoms. On the contrary, septic retinitis presents no inflammatory symptoms.

DIAGNOSIS.—The diagnosis of septic retinitis must rest not alone on the ophthalmoscopic changes, but largely on the general history of the patient. Similar retinal changes, particularly hemorrhages, are found in many general diseases.

TREATMENT must be directed to the removal of the cause. Tonic and supportive measures will be required.

RETINAL SCLEROSES.

Retinitis Proliferans (Proliferating Retinitis; Hyperplastic Proliferating Retinitis).—In this disease numerous bluish-white or gray masses of connective tissue develop in the retina and extend into the vitreous humor.

In a strict sense the affection is not a retinitis, but a proliferation of the connective tissue of the retina. The new tissue obscures the optic disc and often is disposed in interlacing bands. The cause is to be found in repeated and extensive hemorrhages into the retina and vitreous humor, the masses of unabsorbed blood producing atrophy by pressing on the retina and causing proliferation of connective tissue. The masses of newly formed tissue may follow the general course of the retinal vessels, some of which lie under and others over the mass. There is often a development of new vessels in these cases.

Etiology and Treatment.—The etiology of proliferating retinitis is unknown. Trauma and syphilis are supposed factors. The disease is rarely seen. Schöbl observed 2 cases in about 60,000 patients. The prognosis is unfavorable, blindness being the ultimate result. Treatment includes the use of mercury and iodid of potassium by the mouth or mercury by inunction.

Fig. 330.—Proliferating retinitis. (Jaeger.)

Striate Retinitis (Retinitis Striata; Chorioidoretinitis Striata).—In this condition the retina shows yellowish-white or grayish lines which may run in almost any direction, but often pass from the periphery toward the disc. The lines lie beneath the retinal vessels and frequently are branched. They are sometimes found in spontaneously cured cases of retinal detachment. Their cause is not known. Caspar considers the disease the final stage of reattached retinæ, while Holden regards it as due to changes following retinal hemorrhages. Vision usually is much reduced in these cases. The condition does not admit of treatment.

Pigment Streaks on the Fundus (Angioid Streaks).—A few cases are recorded in ophthalmic literature in which the fundus presented long, irregular pigment streaks of a red-brown color, lying beneath the retinal vessels, varying much in size and ramifications, and in their course resembling a system of obliterated blood-vessels. The striæ are of varying diameter, pre-

senting irregular borders and bending sharply. In the cases thus far reported both eyes have been affected. While the disease has been known to follow an injury, in several cases there was no history of trauma. It seems reasonable to attribute the angioid condition to retinal hemorrhages, cases having been observed by Pflange, Knapp, de Schweinitz, and Holden. Pflange states that hyperplastic changes occur in Müller's fibres. Vision may be unaffected, or may be considerably reduced in this disease. There is no known method of treatment of avail in these cases.

Pigmentary Degeneration of the Retina (Retinitis Pigmentosa; Pigmented Retina and Chorioiditis).—By this term is meant a progressive bilateral disease of the retina, leading to atrophy and blindness, and characterized by the presence of pigment collections in the superficial retinal layers, contraction of the blood-vessels, and atrophy of the optic nerve.

Etiology.—The disease is found in from 5 to 10 per cent. of deaf-mutes. It is sometimes attributed to consanguinity in the parents. The influence of consanguinity has been overestimated. Macnamara has shown that the disease is not infrequent among the Hindoos, whose religion prohibits intermarriage. It is also hereditary. It is not infrequent in idiots, in epileptics, in the victims of hereditary syphilis, and in families subject to nervous diseases. In many cases, however, the cause cannot be determined. The disease is either congenital or begins early in childhood, although the pigmentation is probably never present at birth. Retinitis pigmentosa is said by Leber to be more frequent in men than in women in the proportion of about 5 to 2. Other congenital anomalies are not infrequently present in cases of pigmented retina.

Symptoms.—The subjective signs of this disease are (1) night-blindness, (2) loss of visual acuity, and (3) contraction of the field of vision. The most noticeable subjective symptom is night-blindness: *i.e.*, visual acuity is disproportionately lowered under reduced illumination. Often a patient who is unconscious of visual defect by daylight must be led at dusk or on entering a dimly lighted room. Night-blindness may be absent, and in rare cases, in which there is retinal hyperesthesia, vision will be best in reduced illumination. Central vision may remain practically normal for a long time, yet perimetric examination will show there is reduction of the field. Usually there is reduction in central vision in proportion to the contraction of the field. In exceptional instances good central vision is retained in the presence of great narrowing of the field. This contraction leads to loss of orientation. The patient sees as through a tube and often knocks against objects. The constant bowing of the head is characteristic. Occasionally color-blindness exists in these cases.

The ophthalmoscopic signs of pigmentary degeneration of the retina include changes in the vessels, in the optic disc, and in the retina. The vessels are contracted and their number is diminished. Their walls are thickened, producing a corresponding reduction in their lumen. Often they are so small as to resemble mere threads. The optic papilla is of a creamy-

patient needs assistance in getting about. Often, however, the disease remains stationary for long periods.

TREATMENT.—There is no cure for this disease. The best results of treatment are obtained in the form which is caused by acquired syphilis. In all cases of retinitis pigmentosa the eyes should be used in moderation and should be protected from excessive light. Errors of refraction should be carefully corrected. The frequent use of weak miotics, the internal or hypodermic administration of strychnia, and the employment of mercuric bichlorid are among the proposed remedies. Electricity in the form of the galvanic current applied to the eyeball has seemingly produced good results in some cases.

INJURIES OF THE RETINA.

Penetrating wounds of the eye having been considered elsewhere (Chapter IX), in this place the effect of concussion injuries of the retina will be discussed. Such injuries may cause edematous swelling and opacity, hemorrhage, detachment, rupture, pigmentation, or traumatic anesthesia of the retina.

Traumatic Retinitis ("Commotio Retinæ"; Edematous Swelling of the Retina; Concussion of the Retina).—Blows on the eye may be followed by edematous swelling and opacity of the retina, the change being situated generally in a part of the retina opposite to the point of impact. There is marked contraction of the pupil, which dilates imperfectly to atropin. There is some episcleral injection and reduction in the acuity of vision. The tension is normal, as a rule. Stephenson, however, has reported a case of concussion of the retina with minus tension. Ophthalmoscopic examination made an hour or two after the injury will show a grayish-white or milky-white cloudiness existing in disseminated spots and not involving the blood-vessels. Soon the spots may coalesce and the whole fundus may be involved. In from twenty-four to thirty-six hours the process is fully developed. Then it begins to decline, the fundus-reflex again becomes visible, and in a few days the eye is of normal appearance. In rare instances a system of fine radiating lines will be found around the yellow spot. The retina is not thrown into folds, and this fact serves to differentiate commotio retinæ from retinal detachment. The prognosis is favorable. There may be coincident retinal hemorrhages, spasm of the ciliary muscle, and transient astigmatism. The treatment will include rest, the use of atropin drops, and the wearing of smoked glasses.

Retinal Hemorrhage from Trauma is easily recognized ophthalmoscopically by the presence of an elongated clot and by the loss in the continuity of the vessel. The treatment will include rest and atropin.

Traumatic Detachment of the Retina is of rare occurrence. It will be mentioned under another head (page 522).

Rupture of the Retina, apart from the existence of laceration of the other ocular tissues, is a rare condition. The rent may occur at any part

white color, usually not the dead white of atrophy. The edges of the nerve are generally ill defined and bordered with pigment, but the presence of pigment-spots on the nerve-head is very uncommon. The lamina cribrosa is usually hidden from view, and the whole disc looks waxy or transparent. The most striking of the ophthalmoscopic appearances is the presence of irregular pigment-spots, which are most numerous at the periphery and resemble bone-corpuscles in shape. They are usually more abundant on the temporal than on the nasal side of the fundus. Often the pigmentation is most abundant along the course of the larger vessels. Early in the disease the pigment-spots are situated in the far periphery, but as time passes they encroach upon the posterior pole. As the process extends the retinal pigment layer becomes decolorized, thus permitting a view of the chorioidal vessels and giving the fundus a peculiar "wainscoted" appearance (Fig. 2, Plate XXI). *Pari passu* with the growth of the stellate pigment-spots atrophy of the retina and disc become manifest. Vitreous opacities are uncommon in this disease. Posterior polar cataract is occasionally seen, but less frequently than in chorioiditis.

Diagnosis.—In typical cases diagnosis is not difficult. The pigment-spots do not always resemble bone-corpuscles, but may be rounded or irregular like the pigment collections found in chorioiditis. That the pigment is in the retina is determined by the fact that the spots cover the retinal vessels. In the pigment accumulations of chorioiditis the retinal vessels can be seen crossing the spots. Atypical cases of retinal disease have been described which present all of the subjective and objective signs of retinitis pigmentosa except that the pigment-spots are absent. A few cases of unilateral retinitis pigmentosa have been described. Exceptionally the pigment-spots involve the macula, while none are found in the periphery of the fundus. A few cases have been recorded of glaucoma in eyes with retinitis pigmentosa. Blessig has reported the histories of nine brothers and sisters in whom these diseases alternated.

Pathology.—The changes are: an atrophy of the nervous structures of the retina, the nerve-fibre layer usually remaining. The supporting connective tissue becomes hyperplastic. In places the pigment epithelium atrophies; in others, it proliferates, invading all layers of the retina, especially the perivascular sheaths. Pigment granules may be seen scattered in the retina and are sometimes found occluding the lumen of a small sclerosed vessel. Where there is an atrophic area in the pigment layer, and sclerosis of the chorioidal vessels is present, the retina will become adherent to the chorioid. Drusen formations are a frequent accompaniment. Angiosclerosis is more pronounced in the arteries than in the veins, the smaller vessels often being occluded by hypertrophy of the vessel-walls, many of which show hyalin degeneration.

Prognosis.—The prognosis of this form of retinal disease is unfavorable, most of the cases ending in blindness by the time middle life is reached. While total blindness is not the rule, vision is so much reduced that the

PLATE XXI.

Diseases of the Retina.

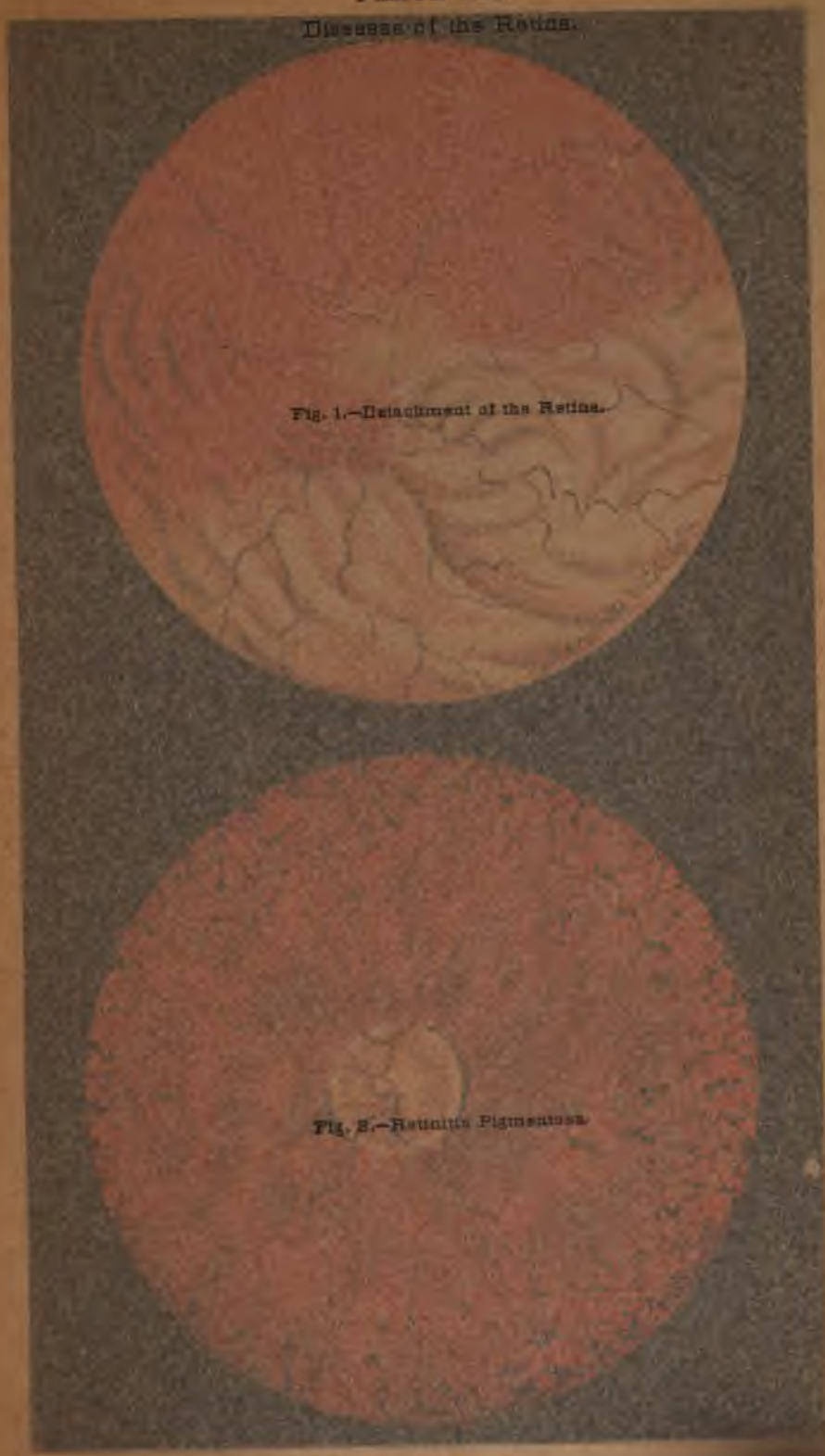


Fig. 1.—Detachment of the Retina.

Fig. 2.—Retinitis Pigmentosa.

DETACHMENT OF THE RETINA.

PLATE 21

Pathology.—Leber noticed that in idiopathic detachment a perforation frequently exists in the floating retina. From ophthalmoscopic findings, pathologic studies, and experiments made on the lower animals, he announced the theory that retinal detachment in non-traumatic cases is due to shrinking of a diseased vitreous humor. This shrinking causes a rent in the retina, and the fluid, which is always found behind the vitreous humor in cases of detachment of this body, passes through the tear in the retina and serves by gravity further to increase the separation of this membrane from the chorioid. Leber's view has been confirmed by Nordensen, who, in 119 cases of detachment, found laceration of the retina in 49 (38.6 per cent.). The latter authority holds that the primary disease is located in the ciliary body and chorioid; this is followed by fibrillary degeneration and subsequent contraction of the vitreous; the vitreous becomes adherent to the retina, and in contracting pulls the retina from the chorioid. The separation of the retina from the chorioid may begin at any part, but probably commences most frequently in some part of the upper half. At the ora serrata and optic disc there is no separation except in rare cases of severe trauma. Thus, the detachment, when complete, assumes a funnel shape; however, the area, depth, and situation of the detachment are subject to great variation. The pigment layer always remains on the chorioid.

In opposition to the views of Leber and Nordensen, Raehlmann has advanced the "diffusion theory," which ascribes retinal detachment to exudation from the chorioidal vessels of a fluid more albuminous than the vitreous humor. He contends that diffusion occurs through the retina, the ingredients of the vitreous humor collecting behind the retina and lifting it up. The diffusion theory has recently found confirmation in experiments made on rabbits by Sinclair. Which of these theories is correct must be determined by future investigations.

Old cases of retinal detachment often show cataract, and the opaque lens frequently undergoes calcareous degeneration. The tension of the eye is usually minus. Both eyes may be involved in retinal detachment; in the majority of cases but one eye is affected.

Symptoms.—Prodromal symptoms of retinal detachment are periodical dimness of vision, photopsiæ, and the appearance of sparks, dust, or soot before the eyes. The subjective signs include a limitation of the field of vision, the appearance of a cloud or of floating specks before the eye, etc. Patients often complain that they can see only a part of an object. So long as the macular region is not involved central vision will be good. Generally patients with retinal detachment will notice sudden loss of vision, but the detached retina may functionate for a time, producing vertigo and metamorphopsia. In uncomplicated cases there is no pain.

Externally there are no noticeable signs. The anterior chamber may appear deeper than normal. The tension is usually minus. Ophthalmoscopic examination shows the normal fundus-reflex lost wholly or in part, the retina in its detached portion appearing as a gray, vibrating, rounded

membrane on whose folds retinal vessels are visible. The vessels are small and of dark color, having lost their central light-streak. The optical condition of the detached retina is that of a highly hypermetropic eye, a strong convex lens being required for the study of its details. The irregular folds of the detachment show white lines,—produced by reflection from the apices of the folds,—while the furrows appear dark (Fig. 1, Plate XXI). The ophthalmoscopic picture is so striking that when once seen it should not be forgotten. Failure to find an old detachment can occur only through opacity of the cornea or lens, or by neglect to examine the periphery of the fundus. Often the detached and floating retina can be seen by ordinary focal illumination. It may be seen to undulate in response to the ocular movements.

Diagnosis.—While recognition of the presence of a detachment of the retina is ordinarily easy, in exceptional instances it is difficult, especially where the detached portion remains unchanged in color. This occurs particularly in recent cases, in young persons, where the fluid behind the retina is clear. In such cases a diagnosis will rest on the determination whether the vessels are smaller, more tortuous, and darker than elsewhere, and on the finding of differences in the refraction of different parts of the fundus. By direct ophthalmoscopy the detached portion can be clearly seen only through a weaker concave or a stronger convex glass than is required for the remainder of the fundus. In indirect ophthalmoscopy the surgeon will need to withdraw his head and the interposed convex lens more or less. Parallactic movement of the vessels over the fundus is often observed in detachment. In many instances careful examination by the direct method will show a tear in the detached retina through which the chorioid is visible. A flat detachment—*i.e.*, one with a thin layer of fluid beneath it—may escape detection. Here the red color of the fundus is intermerged with a grayish cloudiness of the affected area and the vessels are unduly black and tortuous. In old cases of detachment, where the retina is atrophic and has again become clear, the chief diagnostic sign will be the vascular changes mentioned above.

The diagnosis between tumor of the chorioid and simple detachment of the retina has been considered on page 416. Subretinal cysticercus has been mentioned elsewhere in this chapter. Edema of the retina may be mistaken for detachment. In the former condition the fundus presents a more uniform appearance and the retina is not folded. Retinitis proliferans is often associated with retinal detachment.

Prognosis.—The prognosis in detachment of the retina is very bad. Only in rare instances is there a permanent reattachment, spontaneously occurring or following treatment. According to Schöbl, the most favorable cases are those in which detachment occurs in the albuminuric retinitis of pregnancy, in chorioiditis, and in those which follow diseases of the orbit and traumatism. In detachment following myopia the prognosis is unfavorable.

Treatment.—The treatment of retinal detachment is an unsatisfactory —in fact, almost hopeless—task. While in a few rare instances the retina has become reattached spontaneously, and a few recoveries have followed prolonged rest on the back, with hypodermic injections of pilocarpin and the administration of saline purgatives, and some cures have followed the internal use of mercury, iodid of potassium, and salicylic acid, the majority of successful results thus far reported have been attributed to surgical intervention. Surgical intervention, proposed by Sichel in 1859, has assumed numerous forms: simple puncture of the sclera and chorioid (Sichel), discission of the retina (von Graefe), drainage by a fine gold wire passed through the sclera and chorioid (Grizon, de Wecker), stitching the retina to the chorioid by means of catgut (Galezowski), dislaceration with two needles (Bowman), iridectomy (Galezowski and others), injection of iodin into the subretinal space (Galezowski, Gelpke, Schöler), electrolysis (Gillet de Grandmont), cutting of vitreous bands and transfixion of the eyeball (Deutschmann, Jaencke), injection of a 3.5-per-cent. strength solution of gelatin in a physiologic salt solution between the sclera and capsule of Tenon (de Wecker), puncture of the eyeball with the galvanocautery (Galezowski, Abadie), injection of normal salt solution into the vitreous after evacuation of subretinal fluid (Walker), and injection of air into the vitreous (Jensen). Most of these procedures should be ruled out of the domain of modern ophthalmology. All are dangerous to the integrity of the globe, and one of them—intra-ocular injection of iodin—has been followed by meningitis and death.

Of the surgical measures mentioned above, the most promising are simple puncture with a cataract-knife, puncture with the galvanocautery, and subconjunctival injections of salt solution. The last of these procedures has the advantage of innocuousness. Stärkle, of Basel, has reported 23 comparatively recent cases of detachment treated by injections of salt solution, the strength being increased from 2 to 10 per cent. He reports improvement in 21 cases and complete reattachment in 3. Dor, of Lyons, claims 14 complete recoveries in 21 cases. He used subconjunctival injections of 20-per-cent. strength salt solution, leeches, and punctate cauterizations of the sclera, each of these procedures being used once weekly in rotation. Stillson, of Indianapolis, has saved 4 out of 5 cases of detachment treated by the galvanocautery. Winselmann has had 3 cures following subconjunctival injections and the use of compress bandages. While these reports are highly encouraging, it must be remarked that in some of them not sufficient time has elapsed to enable judgment to be passed upon the value of the treatment. Sutphen, however, cured a case of bilateral detachment by scleral puncture and eleven years later vision was normal. Hann and Knaggs have observed a case of symmetrical retinal detachment occurring during labor and associated with albuminuria, with complete recovery. While restoration of vision is more likely to occur in recent than in old cases, some remarkable cases of reattachment are reported. Thus, Dor is

said to have observed reattachment with restoration of vision after seven years, and Wolfe, of Melbourne, had a successful result after three years. Detachment caused by cysticercus and by intra-ocular growths will call for treatment proper for these conditions, viz.: removal of cysticercus and enucleation in tumor. Finally, it must be stated that retinal detachment is only a symptom, and search should be made for the underlying cause.

FUNCTIONAL DISEASES OF THE RETINA.

Hyperesthesia of the Retina (Irritation of the Retina).—This term is applied to a condition in which, without demonstrable ophthalmoscopic lesions, the retina is unduly sensitive to light. Photophobia, neuralgia, blepharospasm, lacrimation, and inability to use the eyes for moderate periods are prominent symptoms. In pronounced cases there is pain in the temples or over the brow; quivering and unsteadiness in vision may be noticed; nausea and dizziness may be present; and, in short, the condition simulates asthenopia from an error of refraction. Yet these symptoms sometimes occur in eyes with normal refraction (Loring). Ophthalmoscopic examination may show veiling of the optic disc, particularly on its nasal side; slight redness of the nerve-head; a striate condition of the retinal fibres passing from the disc; and the whole fundus may appear indistinct. The chorioid may present evidences of hyperemia (page 420).

Etiology.—Hyperesthesia of the retina may be a forerunner of serious organic optic-nerve disease (Loring). Errors of refraction or of muscle-balance and disease of the nasopharynx are causes.

Diagnosis, Prognosis, and Treatment.—The diagnosis of retinal hyperesthesia must be determined by exclusion. The prognosis should be guarded. While the affection does not lead to blindness, the inability to use the eyes may continue for a long period in spite of careful treatment. In the treatment the surgeon should investigate the general condition of the patient most thoroughly. The blood and urine should be examined. The condition of the nose, throat, lungs, etc., as well as the state of the refraction and muscle-balance should be determined. Any departure from the normal should be corrected. Tonics, alteratives, and strychnia may be of benefit in some cases, while others may respond to electricity. Rest, change of scene and of occupation should be considered in intractable cases.

Anesthesia of the Retina (Neurasthenic Asthenopia; Hysteric Amblyopia).—This term is applied to a complicated neurosis in which reduction in acuity of vision and contraction of the visual field are present in connection with functional disturbances in other parts of the body, without the existence of ophthalmoscopic signs of disease.

Etiology.—The causes of anesthesia of the retina are numerous, and include overwork of the eyes or body, lesions of the genito-urinary organs, traumatism, conjunctivitis, errors of refraction or of muscle-balance, intranasal disease, impaired nutrition, prolonged exposure to cold, etc.

SYMPTOMS.—Among the prominent symptoms are inability to use the eyes for ordinary work, irregular variations in the acuity of vision, sudden obscuration of vision, hallucinations of sight, a feeling of heaviness or weariness in the eyes and eyelids, a lack of fixity of the memory-images of observed objects, photophobia, and a diminution or an absence of these symptoms at dusk or on wearing dark glasses.

The perimetric symptoms, however, are of much more value, and include concentric contraction of the field of vision and rapidly occurring variations in the extent and shape of the fields. If two or three perimetric examinations are made in rapid succession, these variations will become apparent. They include contraction or overlapping of the field or reversal of the color-fields. The field may even be hemianopic or defective in sectors, or an oscillating field may be found in which an object recognized at one meridian is soon lost and again regained. A relative central scotoma is not uncommon.

DIAGNOSIS.—The diagnosis of retinal anesthesia must be made by exclusion. Careful ophthalmoscopic examination will fail to show fundus changes. The fields of vision are practically typical of the condition. The loss or obscuration of vision appears and disappears suddenly. It is usually greatest when sympathizing friends are present.

PROGNOSIS.—The affection is not serious, but may be of long duration.

TREATMENT.—The treatment of retinal anesthesia must be conducted on a broad basis. Not only should attention be given to the correction of any ocular abnormality present, but the dietetic, hygienic, mental, and moral conditions should be considered. The rest cure, change of scene and surroundings, exercise, tonics, sea-bathing, and outdoor life are among the measures from which benefit may be expected. The use of dark glasses will be advisable, although some patients become too dependent upon them.

Tuberculosis of the Retina.—Few cases of this disease have been reported. In that observed by O'Sullivan and Story a woman, aged 21 years, complained of loss of vision in the right eye. Ophthalmoscopic examination showed an intense papillitis, the swelling being of a brilliant whiteness. Small, white spots were present in the macular region. Two months later vision was reduced to perception of light, pericorneal injection and discoloration of the iris were present, but the tension was normal. The eye was enucleated, and presented a tumor around the optic-nerve head. Microscopic examination showed typical tubercular structure. The tumor was separated from the chorioid by a coagulum.

CHAPTER XVI.

DISEASES OF THE OPTIC NERVE.

THE optic nerve is liable to congenital malformations, tumors, inflammations, injuries, and functional disturbances.

CONGENITAL ANOMALIES.

Absence (Aplasia) of the Optic Nerve has been observed in a few instances, chiefly cases of anophthalmos. The nerve may be represented by a fibrous cord, which is destitute of nerve-fibres. Rarely its absence exists with the absence of the chiasma and with various anomalies of the nerve-centres.

Atrophy of the Nerve is a rare congenital condition, and is found in cases of microphthalmos and in anophthalmos. Congenital atrophy, the other portions of the globe being normal, has been described.

Coloboma of the Optic Nerve.—In ophthalmic literature about fifty cases are recorded of coloboma of the optic disc, existing either alone or associated with coloboma of the sheath of the nerve. The condition, which was for a long time confounded with the posterior sclerochorioiditis of myopia, is characterized ophthalmoscopically by an apparent increase in the diameters of the optic disc and by the presence of an excavation (Fig. 331). The excavation may surround the disc, but in the majority of cases it is situated inferiorly, which differentiates it from the external (temporal) staphyloma of myopia. The coloboma is generally of a uniformly white or bluish-white color, and sometimes is bordered with pigment. It may assume a funnel shape. In such a case, observed by Randall, the greater part of the disc presented a depth of 6 dioptres, while a circular pit in the lower part of the disc was 4 dioptres deeper. The vessels may emerge in the centre of the disc, but usually they appear at the periphery of the excavation. Coloboma of the disc and sheath may exist separately, together, or in conjunction with coloboma of the chorioid. The position and shape of the coloboma, the irregular position of the vessels, and the frequent existence of other anomalies will serve to distinguish coloboma of the optic nerve from posterior scleral staphyloma. The anomaly is attributed to non-closure of the cleft, which in early fetal life is found in the lower part of the nerve. The so-called *inferior conus*, a condition in which the nerve-head does not fit accurately into the chorioidal aperture, has been found by Wollenberg and Vossius in about 1 per cent. of ophthalmic patients. According to the researches of Jaeger, Schnabel,

and Fuchs, it is to be regarded as a rudimentary coloboma of the sheath of the optic nerve or of the adjacent chorioid.

Eyes showing optic-nerve coloboma often present other defects (microphthalmos, lenticular opacities, lenticonus, persistent hyaloid artery, retinitis pigmentosa, persistent nerve-fibres in the retina, astigmatism, defects in the visual field, etc.). Treatment is limited to the correction of errors of refraction.

Fig. 331.—Coloboma of the optic nerve and chorioid. (Posey.)

Congenital Cupping and other Conditions of the Disc.—These conditions are of rare occurrence. *Congenital cupping of the disc* may show an unusually deep physiologic excavation, suggestive of the glaucomatous cup. The fact that the cup does not extend to the edge of the disc, and the absence of arterial pulsation, will serve to distinguish this congenital abnormality from glaucoma. *Pigment patches on the disc* are often seen, the pigment of the chorioid being continued into the lamina cribrosa. This congenital

condition should not be confounded with pigment-spots acquired after hemorrhage. A black area of pigment with serrated edges, occupying the centre of the nerve-head, was found by Liebreich in *cyanosis oculi*—a condition in which the pigmentation of the whole fundus is markedly increased, the macular region appearing almost black.

The congenital condition known as *spurious optic neuritis* is mentioned elsewhere in this chapter (page 541).

TUMORS.

Primary Intradural Tumors of the Optic Nerve (Fibromatosis Nervi Optici—Byers).—The true, or intradural, primary tumors of the optic nerve are rare, Byers (1901) having been able to collect accounts of only 102 cases in the literature.

ETIOLOGY.—Age is a factor in this disease. Of 85 cases collected by Byers in which the age was recorded, 67 occurred at fifteen years or younger; 32 cases occurred between the first and fifth years. The disease is found more frequently in females than in males, and more often on the left side. Trauma and febrile disturbance or infectious disease are apparent etiologic factors.

SYMPTOMS.—The most striking symptom is the gradual development of painless exophthalmos, the direction of the proptosis being in the majority of cases directly forward, or forward, downward, and outward, although it may be forward and upward, outward, or inward. Exceptionally the exophthalmos has developed rapidly. The patient may complain of pain throughout the distribution of the fifth nerve. The proptosis is attributed chiefly to the direct influence of the tumor, although, in some instances, it is influenced by the state of the orbital blood-vessels, or by stasis in Tenon's space and in the supravaginal lymph-space of the nerve.

A second symptom of importance is the early and great loss of vision. In 69 per cent. of the cases tabulated by Byers vision was absolutely lost in the affected eye at the time of first examination. Variations in visual acuity have been recorded in individual cases.

The ophthalmoscopic changes are various. Of 82 cases in which mention is made of the condition of the fundus, 3 showed simple atrophy of the optic nerve, 34 presented optic neuritis, and 36 gave evidence of post-neuritic atrophy. In 3 cases the fundus was normal. Among the ophthalmoscopic appearances rarely found in primary tumors of the nerve are dilation of the retinal veins, partial detachment of the retina, and hemorrhages. The majority of patients present no lesion of the orbital muscles, although strabismus sometimes is noted, and, indeed, may precede the exophthalmos. In some cases palpation will enable the surgeon to determine the presence of an intra-orbital growth which is not adherent to the orbital walls. The general appearance of the patient does not usually differ from the normal; but cerebral symptoms—such as convulsions and epileptic seizures—have

been noted in rare instances. Dizziness, vertigo, and tinnitus aurium are rarely present. As regards the eyeball, tension is usually normal, but may be minus or plus. A characteristic symptom is antero-posterior flattening of the globe from pressure of the tumor posteriorly, causing the eye to become hypermetropic. Lagophthalmos and keratitis have also been found present.

PATHOLOGY.—The dural covering of the nerve forms a capsule, one-half to one millimetre in thickness, which envelops the tumor. The growth may vary in size from a slight enlargement of the optic nerve to a mass the dimensions of a goose-egg. Usually a piece of normal nerve separates the tumor from the eyeball. In the opposite direction, however, the growth may extend up to or through the optic foramen, and involve the brain. The microscopic diagnosis of the reported cases shows a large number of different terms, the majority being set down as myxosarcomata, myxomata, myxofibromata, or sarcomata. Since tumors of the optic nerve show, in one and the same specimen, several phases of developing connective tissue, Byers considers that they should all be classed as fibromata. When cerebral symptoms occur, and death ensues after removal of a primary tumor of the optic nerve, the result is to be attributed, not to recurrence, but to the continued growth of the intracranial portion of the neoplasm, which could not be removed by operation.

DIAGNOSIS AND PROGNOSIS.—The symptoms enumerated above will enable the surgeon to assert the presence of a growth connected with the optic nerve, but it is doubtful if intradural growths can always be differentiated from extradural ones. The prognosis is serious. The eye in many instances must be sacrificed, and in some cases there is a continued development of the intracranial portion of the tumor, which could not be removed at the time of operation. Where the tumor is located chiefly in the anterior portion of the optic nerve, total removal is feasible.

TREATMENT.—Since the condition rarely shows a tendency toward malignancy, and because in most cases the neoplasm does not encroach on the globe, modern ophthalmologists have sought to extirpate the growth while preserving the eyeball. Scarpa in 1816, Critchett in 1852, and Knapp in 1874 were the first to follow this method. Knapp, in operating on an extradural tumor, made his opening through the conjunctiva and Tenon's capsule between the superior and internal recti, separated the optic nerve from the globe, then cut the nerve at the optic foramen, and pried the tumor out with scissors. Gruening, of New York, was the first to remove a primary intradural tumor of the nerve with preservation of the globe. In recent years Krönlein has devised an operation which is suitable for these cases (see page 652).

Primary Extradural Tumors of the Optic Nerve are ophthalmic curiosities, 18 cases having been recorded. Of these 8 were endotheliomata. This type of tumor generally begins before the age of ten years. Exophthalmos is the most prominent symptom. The loss of vision is slower than

with intradural tumors. The growth increases slowly, is of relatively low malignancy, and causes neither metastasis nor glandular involvement. In none of the recorded cases was the globe invaded (Parsons). The treatment is removal.

Hyalin Bodies in the Nerve-head (Colloid Bodies; Verrucosities; Drusenbildungen).—A rare condition is the presence in the optic-nerve head of clusters of globular, glistening bodies, of a gray or blue-gray color. Attention was first called to these bodies by Müller and Iwanoff, who found them in sections; later observers have noticed the condition ophthalmoscopically. They appear as spheric bodies imbedded in the optic disc or bordering its periphery. They are translucent and have been compared to "half-soaked grains of tapioca" (Gifford). To direct ophthalmoscopy they appear two

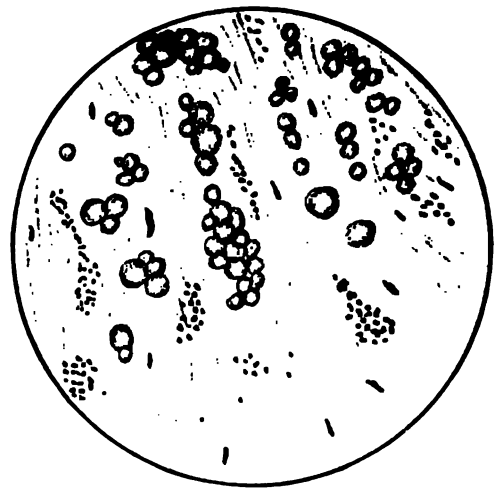


Fig. 332.—Microscopic section showing hyalin bodies in the nerve-head. (Author.)

(Original drawing by Dr. Carl Fisch.)

or three millimetres in diameter, and are best observed by throwing the light to one side of them (Liebreich). In rare instances the hyalin masses may cover the nerve-head, as in the case recorded by Nieden (Fig. 333). The disease may occur in eyes which are otherwise entirely normal, or in association with albuminuric retinitis, retinochorioiditis, or retinitis pigmentosa. As regards the etiology of the affection, Cirincione says: "The first cause of this hyalin transformation and subsequent calcareous deposit in the papilla, however, remains to be determined." Colloid formations found in the nerve-head are quite distinct from the excrescences which spring from the lamina vitrea of the chorioid. While hyalin bodies are more frequent among elderly persons, they have also been observed in children. There is no treatment for the disease.

INFLAMMATION OF THE OPTIC NERVE.

The optic nerve may become inflamed in any part of its course. It is customary arbitrarily to class its inflammations under two divisions: (1) inflammation of the nerve-head, known also as papillitis, intra-ocular optic neuritis, or choked disc; and (2) inflammation of the nerve behind the eyeball, known as retrobulbar optic neuritis. It is evident that in the first form the pathologic changes can be studied by ophthalmoscopy, while in the second type the existence of the disease must be inferred from the subjective symptoms.

Intra-ocular Optic Neuritis (Choked Disc; Papillitis; Stauungspapille).—This condition is of great importance because of the likelihood of its passing into atrophy, and for the further reason that the presence of

Fig. 333.—Hyalin bodies in the optic disc. (NIEDEN.)

inflammation of the nerve-head is of value in the diagnosis of certain diseases of the brain and of the blood. Generally the affection is bilateral.

SYMPTOMS.—Intra-ocular optic neuritis may show no symptoms aside from the ophthalmoscopic signs. Often vision will be normal or only slightly reduced; but it may be markedly impaired. The external appearance shows nothing that is characteristic of the condition. If complete blindness ensues, the pupil will be dilated and the iris will be immobile. Visual acuity may be normal, slightly or much reduced, or abolished. If the macular bundle of nerve-fibres is involved, vision will be seriously affected. The field of vision may present peripheric contraction, sector-like defects, enlargement of the blind spot, scotomata, or hemianopsia. The fields for red and green are generally lost before those for other colors. Color-perception may be defective in persons who present no loss of visual acuity and no limitation of the form-fields. It is evident that little dependence

can be placed upon the subjective symptoms. The diagnosis must rest on the findings of the ophthalmoscope.

The ophthalmoscopic appearances (Fig. 1, Plate XXIII) include hyperemia with edema of the nerve-head; obscuration of the outlines of the disc; the presence of a papillary swelling of the optic-nerve head of greater or less degree; hemorrhages; and dilation of the retinal veins, the arteries being of normal calibre or contracted. The swollen nerve-head, with its numerous distorted vessels, makes a characteristic picture.

The Nerve-head may present only a slight hyperemia, slight swelling, and blurring. In pronounced cases the swelling forms an elevation of several dioptres' height. The elevated area is of a grayish-white color, is clouded, and presents radiating striæ. It gradually runs into the surrounding retina. The boundaries of the nerve-head are obliterated and the location of the papilla becomes known only by the convergence of the vessels. The hyperemia, which is often intense, may be confined to the area of the swollen papilla or may also involve the adjacent retina. Hemorrhages are often seen in the swollen tissue. In cases of intracranial growths which have produced double (bilateral) optic neuritis, attempts have been made to distinguish cerebral from cerebellar growths by the appearance of the swollen disc. Oliver states that in subtentorial (cerebellar) tumors the disc looks denser and harder than in cases in which the tumor is situated in the cerebrum.

The Vessels in papillitis are tortuous and resemble a bundle of angle-worms. The veins are dilated, tortuous, and dark. The arteries are tortuous, and are either of normal or of diminished calibre. Both arteries and veins are partly concealed by the swollen tissue. The light-streak is present. The adventitia may be thickened, causing the appearance of white lines along the vessels. The tortuosity of the vessels makes a picture which has been compared to the head of Medusa.

Hemorrhages, which are usually small, are not uncommon. They may be limited to the area of the swollen papilla or may involve the retina as well. They may assume various forms. When existing in the nerve-fibre layer of the retina, they are of flame-shape. Only a single hemorrhagic spot may be present or there may be numerous such areas.

Types of Intra-ocular Optic Neuritis.—Early in the history of ophthalmoscopy the inflammations of the nerve within the globe were classified as (1) *descending neuritis,* (2) *choked disc,* and (3) *neuroretinitis.*

1. Von Graefe, as the result of the ophthalmoscopic study of cases of intra-ocular optic neuritis occurring in meningitis, assumed that the morbid process extended from the cerebral meninges along the course of the nerve. Hence the term *descending neuritis.* This condition is characterized by moderate swelling of the nerve-head, slight discoloration of the disc, slight changes in the vessels, and the presence of an exudation, which causes opacity of the papilla. The process tends to extend to and involve the retina.

2. In cases presenting marked swelling of the disc, hemorrhages, and signs of vascular distension, von Graefe applied the term *"Stauungspapille,"* which, in English, is *choked disc.*

3. Cases presenting areas of hemorrhages along the course of the retinal vessels, and areas of degeneration in the retina, together with blurring of the edges of the optic disc, or marked swelling of the same, are known by the name *neuroretinitis.* This is a frequent condition in renal diseases.

4. The preceding terms (Nos. 1 and 2) carried with them disputed and confusing ideas as to causation. Hence Leber proposed the term *papillitis,* to include all the inflammatory changes occurring in the intra-ocular end of the optic nerve.

5. Monocular neuroretinitis is of rare occurrence. It may result from orbital causes (inflammations, traumatisms) or from intracerebral causes. It has been observed in anemic, rheumatic, syphilitic, and pregnant patients; and may result from variola, scarlatina, or diphtheria (Millikin).

DIAGNOSIS.—While generally easily determined, if the media are clear the diagnosis of intra-ocular neuritis may present many difficulties. The signs of greatest value are (1) obscuration of the edges of the disc, (2) swelling of the nerve-head, and (3) increased redness or change in color. Of these, the third is of little value. *Redness* may be caused by hypermetropia. If due to eyestrain, it will disappear in a short time under the influence of a mydriatic and the correction of the error of refraction. The disc is also subject to physiologic variations in color. Inflammation may cause the disc to present a lilac-gray tint.

Obscuration of the Disc-margins is a valuable sign. Blurring of the disc from improper focusing may cause the beginner to diagnosticate optic neuritis in an eye which, except for the presence of an error of refraction, is normal. Fine, dust-like opacities in the vitreous humor may produce an obscuration resembling that which is found in the early stages of optic neuritis. These opacities can be seen by the aid of a strong convex lens placed behind the ophthalmoscope, under reduced illumination. Obscuration of the nerve-margins from the presence of hyalin bodies or from opaque nerve-fibres can scarcely be mistaken for neuritis. Tags of connective tissue may obscure parts of the optic disc, forming a thin, gauze-like veil, whitish patches, or delicate shreds. If congenital, they will hide the vessels; if resulting from disease, the vessels will be constricted.

Swelling of the Nerve-head is characteristic of intra-ocular optic neuritis. The difference in level between the retina and papilla can be determined by the parallactic test and by direct ophthalmoscopic examination. The prominence is best seen with the binocular ophthalmoscope.

The congenital condition known as *spurious optic neuritis* may cause mistakes in diagnosis (see page 541).

ETIOLOGY.—Among the causes of optic neuritis are alcoholism, anemia, aneurisms, caries of the adjacent bones, chlorosis, leukemia, cerebral abscess, cerebral hemorrhage, cerebritis, meningitis, epilepsy, chorea, general paraly-

sis, disseminated sclerosis, hydrocephalus, hydatids, lead poisoning, tuberculosis, malaria, rheumatism, gout, syphilis, injuries of the head and spine, myelitis, tetany, loss of blood, orbital diseases, nasal diseases, pertussis, typhoid and typhus fevers, renal diseases, diabetes, pneumonia, suppurative diseases of the ear, errors of refraction, eyestrain, tumors of the brain, tumors growing from the meninges or optic nerve, congenital deformities of the skull, pregnancy, retained secundines, heat-stroke, menstrual disorders, excessive lactation, checking of the lacteal secretion, and heredity. The most frequent cause is intracranial tumor, double optic neuritis occurring in from 80 to 95 per cent. of these cases. All types of morbid cerebral growth are likely to cause papillitis. Second in frequency as an etiologic factor is meningitis. The third place should probably be assigned to syphilis. Multiple sclerosis rarely causes optic neuritis, Uhthoff having found the condition only five times in one hundred cases, and in only three was the neuritis of high degree.

PATHOLOGY.—In cases of pronounced papillitis the swollen nerve-head is seen macroscopically as a mass projecting beyond the retina. The adjacent retina is pushed aside and in neuroretinitis it is thrown into folds. In some cases an ampulliform swelling of the distal end of the optic nerve is present. Microscopic examination shows edema and swelling, hemorrhagic extravasations, and varicosities of the nerve-fibres. There is slight infiltration of leucocytes. In interstitial neuritis the morbid process begins in the sheath and septa. Edema, cellular infiltration, and fibrinous exudation are followed by a new formation of connective tissue. Atrophy of the nerve-fibres ensues and leads to scotomata and sector-like defects in the field of vision.

COURSE.—The disease may be acute or chronic. It may appear suddenly, reaching its greatest development within a few days; or it may come on slowly, lasting for months or even years and be attended with progressive loss of vision. Second attacks of intra-ocular optic neuritis have been reported.

PROGNOSIS.—This must be guarded, since it is impossible to determine, at the first examination, whether the disease will end in blindness, partial loss of vision, or restoration of visual acuity. The cause must be determined, if possible. If due to syphilis, rheumatism, disorders of menstruation, or anemia, the prognosis will be favorable. Intra-ocular optic neuritis which is produced by intracranial lesions offers an unfavorable prognosis.

MECHANISM OF PAPILLITIS.—Many theories have been advanced to account for the mechanism of papillitis.

1. Von Graefe attributed the papillitis which accompanies meningitis to "descending neuritis." The swelling of the nerve-head, which is found in intracranial diseases, he attributed to obstruction of the return of blood from the eye, by compression of the cavernous sinus. This, known as *the back-water theory*, held sway until 1869, when Sesemann showed that the communication between the orbital and facial veins is so free that pres-

sure on the cavernous sinus can produce only transient engorgement of the retinal veins.

2. Stellwag in 1856 and Manz in 1865 showed that the sheath of the optic nerve is distended in optic neuritis produced by brain tumors and meningitis. In 1869 Schwalbe demonstrated that the subvaginal space around the optic nerve is continuous with the subdural space around the brain. The same year Schmidt-Rimpler attributed papillitis to distension of the optic-nerve sheath, thus originating *the lymph-space theory*. Manz, who supported this theory in 1871, attributed the intra-ocular changes to simple pressure on the nerve and blood-vessels. Schmidt-Rimpler sug-

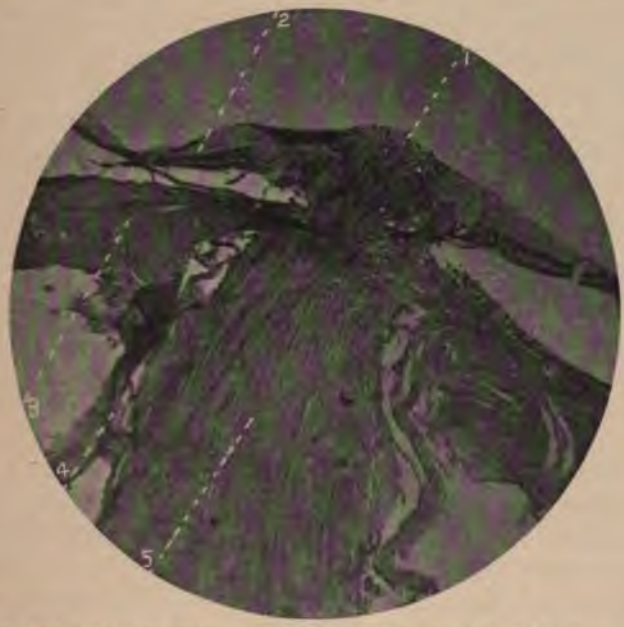


Fig. 334.—Photomicrograph of the optic-nerve head in case of tumor of the brain. (AUTHOR.)

1, Swollen nerve-head. *2*, Retina. *3*, Sclera. *4*, Sheath of the nerve. *5*, Orbital portion of the nerve.

gested that optic neuritis was due to the irritant nature of the fluid distending the lymph-spaces.

3. Leber in 1881 rejected both the "mechanical pressure" idea of Manz and the "irritation" theory of Schmidt-Rimpler. He held that the distension of the sheath is the immediate cause of papillitis, by reason of the conveyance of pathogenic material to the optic nerve behind the eye. This was the beginning of the *inflammatory theory*, which has undergone various modifications. Deutschmann in 1887 accepted it and published the results of confirmatory experiments.

4. Benedikt, Hughlings Jackson, and Brown-Séquard held that an intracranial tumor acts as a foreign body, causes irritation, and produces a

reflex influence (via the vasomotor nerves) upon the optic-nerve head. This, known as *the vasomotor theory*, has been generally rejected because it involves a mechanism which may not exist.

5. Edmunds and Lawford regard the optic neuritis which is produced by intracranial lesions as due to basilar meningitis, the inflammation reaching the nerve-trunk by way of its sheath. *Parinaud's theory* is founded upon the coincidence of internal hydrocephalus and papillitis. Internal hydrocephalus causes a general cerebral edema. The optic nerve participates in the process, the papilla becomes edematous, and the subsequent changes are due to this edema.

6. *The theory of Sourdille* (1901) is founded on the coincidence of internal (ventricular) hydrocephalus and optic neuritis. He calls attention to the fact that the anterior three-fifths of the chiasma are covered only by pia mater and by the visceral layer of the arachnoid, while the posterior two-fifths project into the third ventricle and are covered by ependyma. Edema, originating in the gray matter of the third ventricle, passes by contiguity to the neuroglia of the optic nerves. The nerve, increasing in size, is compressed by the unyielding optic foramen; the return circulation of the blood from the veins of the optic nerve, which enter the cranial cavity, is interfered with, as also is the lymphatic circulation. The interstitial edema of the nerve increases and the veins of the pia mater become dilated. A serous exudate, which accumulates in the subarachnoid spaces, appears. Destruction of the sheaths of the nerves follows, and thus is formed the classic ampullar dilation. The central retinal artery becomes contracted, but it remains pervious. The vein is reduced to a mere slit, and were it not that there developed a communication between the papillo-optic circulatory system and the vessels of the chorioid and scleral ring, vision would be quickly destroyed. The development of the collateral circulation produces those changes which are visible ophthalmoscopically and which are known as "choked disc." The swelling is limited to the nerve-head, and does not involve the retina because of the great compensatory dilation which the vessels, traversing the lamina cribrosa, undergo in performing the extra work thus thrown upon them. Another reason for the limitation of the swelling is that the capillary anastomosis exists only in the region of the nerve-head. Hence the retina remains free of involvement. This capillary anastomosis influences the anatomic changes which are found in the lamina cribrosa. The degree of swelling varies with the permeability of the central retinal vein and with the relative size of the optic foramen. It may appear only as a cloudiness, presenting the appearance which von Graefe denominated "a descending neuritis." Up to this point a cure is possible, provided the cause can be removed. In some cases in which the tumor of the brain continues to grow, vision will be retained for a long period. The swelling of the papilla may diminish, owing to the fact that atrophy of the nerve in the optic foramen renders return of fluid possible; or the improvement may be due to a further devel-

opment of the collateral circulation through the veins of the dural sheath. Vision gradually is lost. Lesions of degeneration appear in the nerve. The peripheral fibres are the first to suffer; the central ones are involved later in the course of the disease. Thus all the phenomena are the result of edema of the ependymal neuroglia, which is an extension of the edema of the gray matter of the brain, found in all cases in the vicinity of growing cerebral tumors.

TREATMENT.—This will depend upon the cause. When present in anemic subjects, intra-ocular optic neuritis generally yields to a course of treatment by tonics. If due to rheumatism, salicin, salicylate of sodium, the iodid of potassium or of sodium, pilocarpin, hot baths, etc., should be employed. When caused by syphilis, active mercurial treatment should be instituted and may be attended with the use of full doses of potassium iodid. Here pilocarpin may be useful. Some authors use these remedies simultaneously, employing mercury by inunction, potassium iodid by the mouth, and pilocarpin hypodermically. If the etiology is obscure, this plan should be followed. The application of cups or of leeches to the temples is used by some surgeons with possible benefit. The eyes should be protected by the use of dark glasses.

Trephining of the skull, and excision of tumors where this is possible, will often be followed by improvement because of the reduction of intracranial pressure. Local surgical measures, such as incision of the distended vaginal sheath, have not met with favor. In a few cases trephining of the ethmoidal and sphenoidal sinuses has been followed by excellent results.

Retrobulbar Optic Neuritis (Orbital Optic Neuritis).—Inflammation of the orbital portion of the optic nerve may occur as an acute or as a chronic process. Owing to the location of the lesion, the ophthalmoscopic signs are either slight or not characteristic. After the neuritis has subsided the optic-nerve head will show atrophy.

ACUTE RETROBULBAR OPTIC NEURITIS.—This, the fulminant form of retrobulbar neuritis, is of rare occurrence.

Etiology.—Among the causes are rheumatism, gout, syphilis, tuberculosis, exposure to intense cold, and infectious diseases (influenza, etc.). It has been seen to follow typhoid fever, carcinoma of the stomach, poisoning by the bite of a scorpion (Gonzales), lead poisoning, and diabetes. Vail has observed retrobulbar optic neuritis in cases of intranasal disease with involvement of the sphenoid bone. The disease may follow facial paralysis, the optic neuritis occurring several months or years after the involvement of the facial nerve. Alveolar abscess, by causing orbital periostitis, may produce the disease. Orbital cellulitis is also a cause. Some patients with disseminated sclerosis show great loss of vision without marked ophthalmoscopic signs, one eye usually being affected. In this disease bilateral involvement is more frequent in the form of chronic retrobulbar optic neuritis.

Symptoms.—The symptoms include sudden diminution or obscuration of sight, beginning in the centre of the field and leading rapidly to great loss of vision or to complete blindness. In some cases the papilla is swollen and both eyes are affected, thus resembling the intra-ocular optic neuritis which is associated with intracranial disease. In such retrobulbar cases the changes in the disc are not gross, and there is an absence of other symptoms of intracranial disease. Orbital pain and tenderness are present in this form of retrobulbar neuritis. However thorough the restoration of vision may be, the optic disc in cases of retrobulbar neuritis shows evidence of the past attack, in the form of pallor of the temporal half of the nerve-head. Blindness may appear in from one to eight days. Headache or dull pain in the orbit is generally present. At first the ophthalmoscope shows nothing pathologic; but later the nerve-head becomes hyperemic, the margins are blurred, the surrounding retina is hazy, and, in rare instances, small retinal hemorrhages and grayish or yellowish spots of exudation appear in the macular area. The arteries may be diminished in calibre, while the veins are enlarged and tortuous. Movement of the eye, or direct backward pressure upon it, will cause pain. Pain is limited to the diseased side and is unaccompanied by vomiting. Occurring in cases of acute or subacute myelitis, there may be marked swelling of the nerve-head. Obviously the line between intra-ocular and retrobulbar optic neuritis is sometimes an arbitrary one. After weeks or months signs of atrophy of the optic disc appear. Usually only one eye is involved. If the second eye becomes affected, there is generally an interval of months. Visual acuity is worse in a bright light. Patients often complain of seeing objects as through a moving haze. Dimness of sight may be complained of where vision is $^6/_6$; and color-scotomata may be found. As a rule, vision is reduced to one-fourth or one-third the normal. With the decline of the acute symptoms, the ophthalmoscopic evidences of optic-nerve atrophy become manifest.

Pathology.—The changes include atrophy of the nerve-fibres, increase of the nuclei, and thickening of the trabeculæ and of the walls of the small vessels. Gunn states that pallor of the optic disc occurring subsequent to retro-ocular neuritis does not necessarily mean atrophy of the nerve-fibres, but may be due to increase of connective tissue secondary to lymphatic obstruction.

Diagnosis.—The disease is to be differentiated from suddenly discovered congenital amblyopia and from hysteric amblyopia.

Prognosis.—Acute retrobulbar optic neuritis runs an acute course, which ends either in complete or in partial recovery. Recovery of useful vision is possible if improvement begins early. Nettleship believes that the affection may be a forerunner of spinal-cord disease. In favorable cases vision will become normal. In unfavorable ones there will be a central scotoma. Since the disease may cause blindness, a guarded prognosis should be given.

Treatment.—Where possible, this must be carried out according to the etiology of the disease. Rest of the eyes and the energetic use of diaphoretics will be advisable. If rheumatism is regarded as a cause, salicylate of sodium should be given.

Chronic Retrobulbar Optic Neuritis (Toxic Amblyopia).—This may follow repeated acute attacks, or the disease may be chronic from the beginning.

Etiology.—Among the causes are rheumatism, gout, exposure to cold, periostitis, meningitis, disseminated sclerosis, systemic poisoning with drugs, and heredity. The cases which are due to poisoning with drugs are classified as instances of *toxic amblyopia* and are discussed elsewhere in this chapter.

Symptoms.—There is gradual reduction in visual acuity, with the presence of a central scotoma. The ophthalmoscopic findings may be negative or there may be pallor of the temporal segment of the optic disc. The symptoms will be more fully considered under "Toxic Amblyopia."

Prognosis.—This is favorable, provided the cause can be removed before atrophy of the nerve-fibres ensues.

Treatment.—This will be the same as that given for toxic amblyopia.

Optic Neuritis with Nasal Discharge.—A few cases have been reported in which there was persistent dropping of watery fluid from one side of the nose (usually the left), preceded or followed by severe cerebral symptoms, and associated with optic neuritis, which led to atrophy and blindness. The cerebral symptoms include headache, vertigo, vomiting, stupidity, epileptiform seizures, sleepiness, delirium, coma, loss of power in the lower extremities, great reduction or complete loss of vision, and papillitis. Both eyes are affected. Analyses of the nasal discharge have not shed much light upon the nature of the disease. In Nettleship's case the fluid was attributed to a diseased nasal mucous membrane, and did not show the characteristics of cerebro-spinal fluid. Priestley Smith, who observed two cases of this rare disease, inclined to the view that disease of the nose or of its accessory cavities is responsible for the cerebral symptoms. Leber has recorded a case of internal hydrocephalus associated with dropping of fluid from the nose. He believes that the fluid had its origin in the third ventricle, escaping through an opening in the ethmoid bone, or from the subdural space via lymph-spaces surrounding the olfactory nerves. The whole subject demands further study. The prognosis is unfavorable both as regards vision and life. Treatment thus far has been without result.

Spurious Optic Neuritis, although it is a congenital anomaly, will be mentioned in this place. The term is used to describe a condition in which the nerve-head is unusually prominent, the disc-margins are blurred, and white lines accompany the unusually tortuous retinal vessels. The disc may be red, giving the appearance of hyperemia; or it may be pale and blurred, resembling a subsiding neuritis. The prominence of the disc is sufficient to permit the expert ophthalmoscopist to measure it. Since these

appearances are observed only in hypermetropic eyes, the term *hypermetropic disc* seems to be appropriate. The degree of error has no influence upon the anomaly. So closely does spurious optic neuritis resemble true inflammation of the nerve-head that the differentiation can be made only by the fact that the former condition remains stationary. Vision in these cases is usually normal after the refraction-error has been corrected. The fields of vision are also normal.

TOXIC AMBLYOPIAS.

Toxic Amblyopia.—This term is applied to cases of dimness of vision which are caused by toxic substances. Among the synonyms are chronic retrobulbar optic neuritis; intoxication-amblyopia; alcohol-amblyopia; quinin-amaurosis; tobacco-amblyopia; malarial amblyopia, etc.

ETIOLOGY.—The agents which are capable of producing toxic amblyopia are numerous. Among them are alcohol, tobacco, bisulphid of carbon, iodoform, stramonium, tea, coffee, chocolate, thyroidin, salicylic acid and the salicylates, lead salts, quinin, oil of wintergreen, filix mas, pelletierin, nitrobenzol and dinitrobenzol, anilin, ergot, the venom of serpents, mercury, phosphorus, sulphur, antipyrin and antifebrin, carbolic acid, cannabis Indica, conium, digitalis, carbonic acid gas, naphthalin, santonin, mescal, strychnin, nitrite of amyl, the mydriatic drugs, gelsemium, curare, cocain, eucain, holocain, benzin, nitroglycerin, etc. The patients are generally males of or above the age of forty years.

SYMPTOMS.—The chief symptom is a disturbance or loss of vision. The patient will complain of "misty," "smoky," or "foggy" vision. He will notice difficulty in reading, and this will cause him to ask the surgeon for glasses or for a change in lenses. At this early stage central vision may be normal or only slightly reduced. Ophthalmoscopic examination will show a pallor of the temporal segment of the nerve-head, with or without slight blurring of the margins of the disc. The nasal side of the disc may be hyperemic. Perimetric examination made at this time will show the presence of negative central scotomata, which are of oval shape and include the blind spots and fixation-points. In this zone there is loss of color-vision. Green is the first color to disappear, and is followed by the loss of red and blue. Finally form-perception is lost in this area.

In advanced cases vision is reduced to $^{20}/_{40}$ or even to $^{6}/_{200}$ for far, and, to the reading of Jaeger, 5 to 14 for near. The temporal segment of the nerve-head is then of a chalky-white color.

PATHOLOGY.—Holden and Nuel have shown that the first change in toxic amblyopia is chromolysis of the retinal nerve-cells. This progresses and the nerve-cells are destroyed. The optic-nerve changes—destruction of nerve-fibres, proliferation of the interstitial connective tissue, etc.—are secondary to the retinal lesions. It seems to be definitely settled that quite diverse types of optic neuritis are of parenchymatous nature. These neu-

ritides are tabetic retrobulbar optic neuritis, infectious microbic optic neuritis, simple white atrophy, and some forms of papillitis. Birch-Hirschfeld states that the pathologic changes in toxic amblyopia have a double origin (in the retina and in the optic nerve), but thinks that the ganglion-cell changes in the retina precede or at least come at the same time as the nerve-fibre degeneration.

Quinin-amaurosis.—Quinin may cause a temporary amblyopia or an amaurosis which presents grave symptoms. While this condition is called quinin-blindness, it may be caused by any of the alkaloids found in the bark or may result from a large dose of the tincture of the bark. The sulphate and bisulphate of quinin are more active than other salts. Owing to the fact that in most of the cases the patients were suffering from general diseases,—such as malaria, syphilis, meningitis, pneumonia, typhoid fever, gastro-enteritis, etc.,—it has been doubted that the blindness was due to the

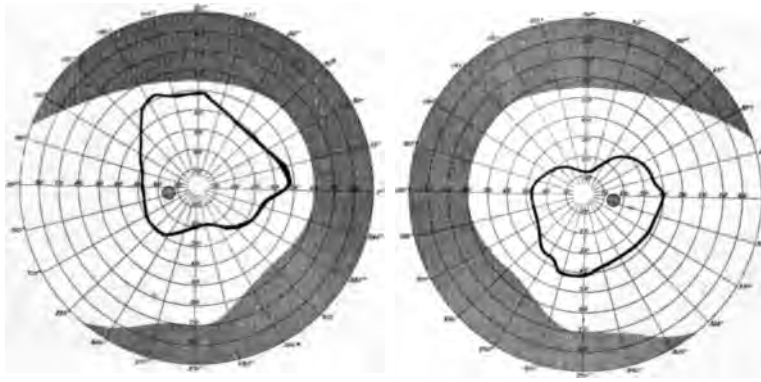


Fig. 335.—Fields of vision in a case of quinin-amaurosis. (AUTHOR.)

Taken two weeks after the ingestion of sixty grains of the drug in twenty-four hours.

ingestion of quinin. On the other hand, the exact clinical picture of quinin-amaurosis can be produced in dogs.

The dose causing blindness has varied from 15 grains to 1 ounce in twenty-four hours. Occasionally temporary blindness has appeared after the administration of 15 grains in divided doses in twenty-four hours. Of 69 cases gathered from literature by de Schweinitz, 42 were males, 20 females, and in 7 the sex was not given. The ages varied from 3 to 65 years. Thirty-four were under 40 years of age. One case is recorded in which a negro became blind from quinin.

The symptoms of quinin-amaurosis are:—

1. Total blindness following the ingestion of large quantities of quinin.
2. Contraction of the visual fields.
3. Marked contraction of the retinal blood-vessels.
4. Pallor of the optic discs.

The pupils are widely dilated and do not contract on exposure to light; but, according to some observers, they contract to a slight extent on accom-

modative effort. This was true of a patient seen by the author. Erythropsia has been observed in one case.

Pathology.—To explain the occurrence of this type of amaurosis we must look to the vasomotor system. In dogs poisoned by quinin de Schweinitz found thickening and changes in the walls of the optic-nerve vessels (endovasculitis), thrombosis of the central artery with organization of the clot, and complete atrophy of the visual tracts. Holden has shown that the toxic effect is exerted primarily upon the ganglion-cells of the retina.

Diagnosis.—Quinin-amaurosis might be mistaken for embolism or thrombosis of the retinal vessels. These affections are unilateral, while quinin-amaurosis is almost always bilateral.

Prognosis is favorable so far as the restoration of central vision is concerned. In grave cases the visual field is not restored in its entirety. In one instance the patient was practically blind two years after the administration of quinin, vision in the better eye being equal to perception of movements of the hand at six feet. It is probable that other cases equally unfortunate have occurred, but have not been reported.

Treatment.—The drug should be discontinued. The patient should be placed in the recumbent position. He should be fed with nutritious substances, such as beef-broth, soups, etc. Inhalations of nitrite of amyl can be cautiously tried. Strychnin should be used by the mouth or hypodermically.

Methyl-alcohol Amblyopia.—Owing to its cheapness, wood-alcohol is coming into extensive use in the arts, replacing ethyl-alcohol. Methyl-alcohol when taken by the stomach, or when inhaled, may cause blindness. Most of the reported cases concerned persons who had become drunk by the use of wood-alcohol or had imbibed freely of a cheap quality of Jamaica ginger. The disease has been observed also among artisans (painters, shellackers) who work with preparations containing wood-alcohol. The poisonous action of the drug is manifested by headache, vomiting, dizziness, and dimness of vision. The pupils will be widely dilated and may react to light, but not to accommodation. The field of vision will be contracted peripherally and will show a central scotoma; and there will be a reduction, of greater or less degree, of visual acuity. In a few days, vision may improve, but the improvement is only temporary. Ophthalmoscopic examination will give different results, according to the time which has elapsed since the intoxication and according to the amount of the toxic substance imbibed or inhaled.

Early in the case there may be no visible changes. Sherer, on the other hand, records a case in which examination made one day after intoxication showed a greenish-white discoloration of both optic-nerve heads, together with atrophic cupping.

Diagnosis.—The history of imbibition of methyl-alcohol or of preparations containing it, or exposure to its fumes, together with such symptoms

PLATE XXII.

Diseases of the Optic Nerve.

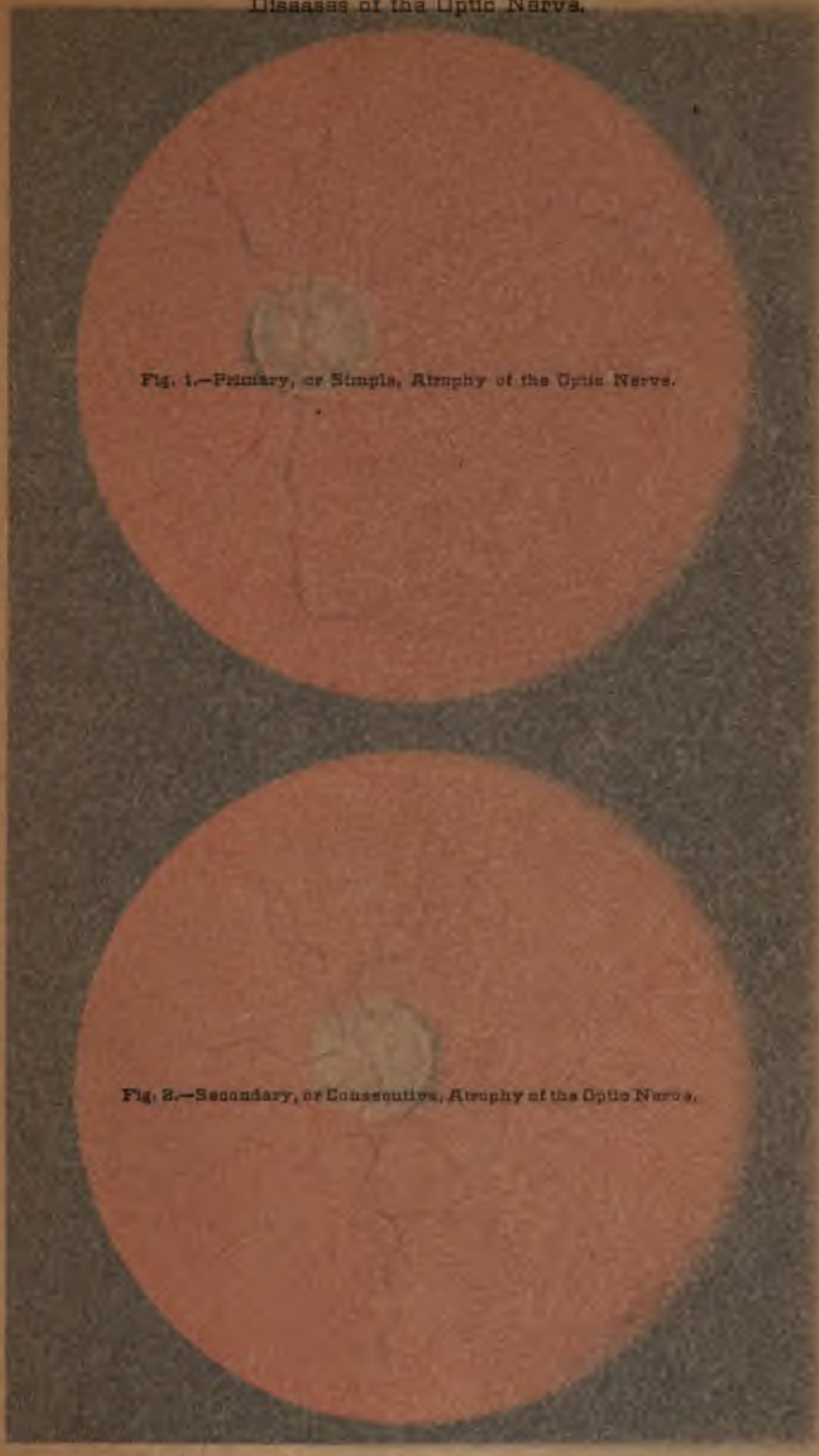
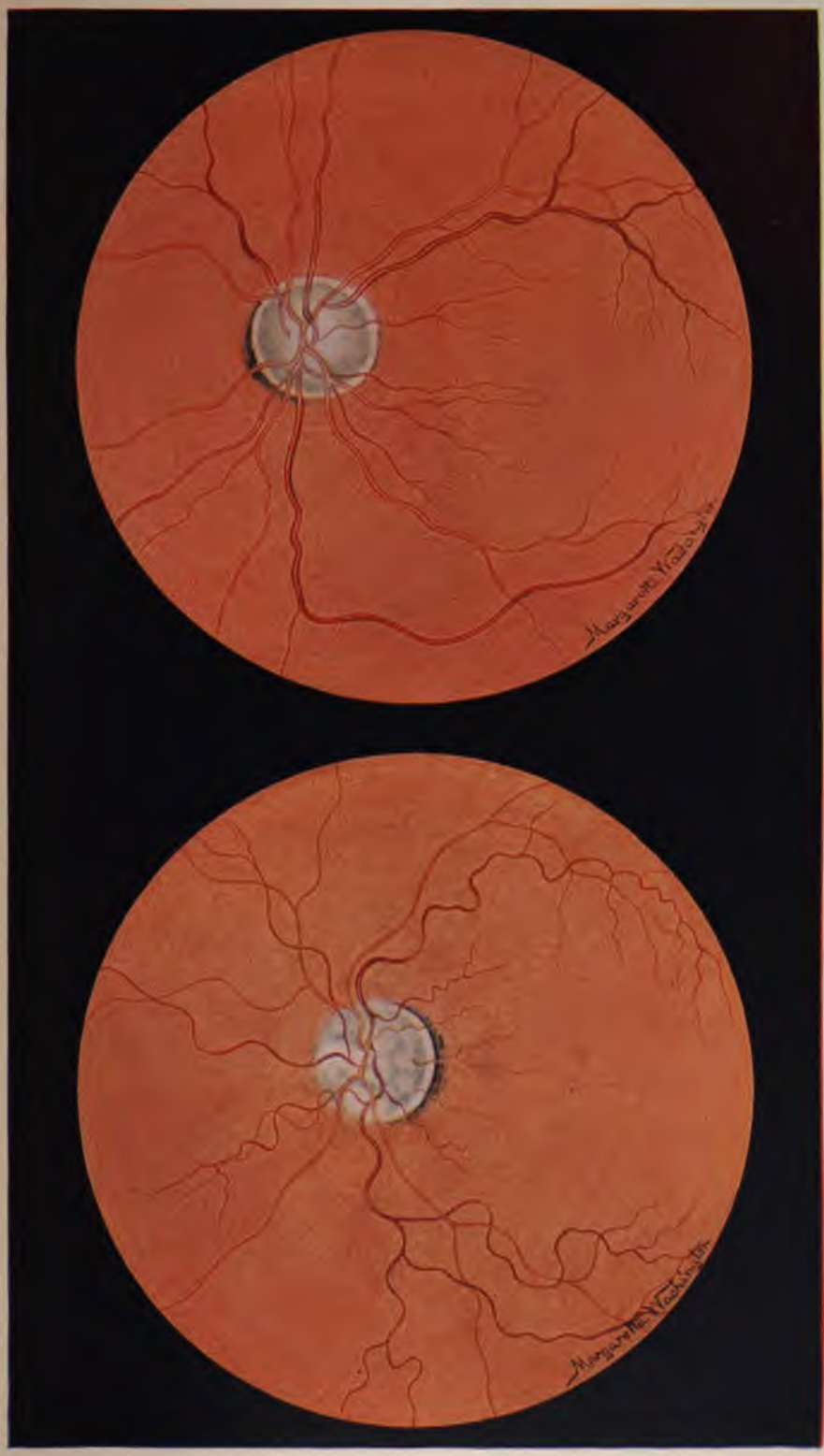
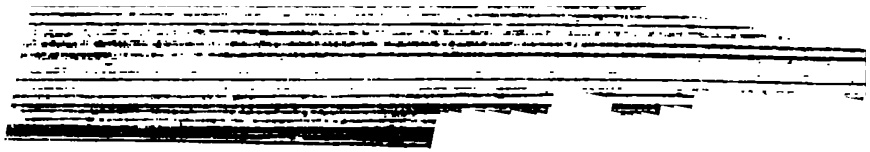


Fig. 1.—Primary, or Simple, Atrophy of the Optic Nerve.

Fig. 2.—Secondary, or Consecutive, Atrophy of the Optic Nerve.

PLATE 22





as headache, nausea, vomiting, dizziness, mental disturbances, and loss of vision, will lead to a correct deduction.

PROGNOSIS must be guarded.

TREATMENT.—This includes pilocarpin sweats and the use of potassium iodid and strychnin.

Ptomain-poisoning (Allantiasis; Botulism).—The toxic substances found in decaying meats, in putrid fish, "high" game, decomposed sausage, etc., cause amblyopia. Other ocular symptoms are paresis or paralysis of the accommodation, ptosis, and paralysis of the extra-ocular muscles. If death does not occur, the ocular symptoms may rapidly disappear. In some cases the paralyses remain and are attributed to nuclear hemorrhages or to basilar meningitis. Treatment will consist of stimulants, tonics, etc.

ATROPHY OF THE OPTIC NERVE.

Atrophy of the Optic Nerve.—The term atrophy of the optic nerve is applied to conditions in which there is a degeneration of the nerve-fibres and an overgrowth of connective tissue. The disease may be primary or secondary.

PRIMARY OPTIC-NERVE ATROPHY (Fig. 1, Plate XXII) develops without previous visible signs of inflammation. As a rule, both eyes are involved, the process being more advanced in one than in the other. The optic disc is shallowed and changed in color, and the disc-margins are clearly defined.

SECONDARY OPTIC-NERVE ATROPHY (Fig. 2, Plate XXII) develops as a result of a previous inflammatory process, which may be located in the brain or its membranes, in the chorioid, in the retina, or in the nerve-head. The disc-margins are blurred by the deposition of connective tissue upon them. The distinctions given above are based on clinical appearances. Pathologic studies have shown that, in many cases, what clinicians call simple or primary atrophy of the optic nerve is due to parenchymatous neuritis (Nuel).

EXTERNAL AND SUBJECTIVE SYMPTOMS.—The external appearance is that of the normal eye, except that in total blindness the pupils will be dilated and irresponsive to light. In some cases, particularly those due to tabes, the pupils are small, reacting to accommodation and convergence, but not to light (Robertson pupil). Pain and photophobia are generally absent. The disturbance in vision is manifest in a lowering of visual acuity, a contraction of the form-field, and a contraction of the color-field.

1. The loss of central vision varies from a slight reduction to complete blindness. Any existing error of refraction should be corrected before deciding that a loss in visual acuity is present. If the atrophy is bilateral, the loss of vision is generally greater on one side. Vision is impaired both for distance and for near.

2. Contraction of the field for white is found at an early date. The common defect is a concentric contraction which progresses until only a

small central vision-area remains. Quadrant-shaped defects, hemianopsia, and central and ring-shaped scotomata have been observed.

3. Contraction of the color-field is an almost constant symptom, the colors being lost in the following order: Green, red, blue, yellow.

4. The light-sense is diminished.

Ophthalmoscopic Changes are found in the nerve-head, in the surrounding tissues of the fundus, and in the blood-vessels.

In *primary atrophy* (Fig. 1, Plate XXII) the margins of the optic disc are well defined. The disc itself presents a stippled appearance, and is of a grayish or bluish color. The surface of the disc is slightly concave, and the physiologic cup is not filled. The vessels are slightly reduced in calibre. The fundus looks normal.

In *secondary atrophy* (Fig. 2, Plate XXII) the disc-margins may be irregular, and are blurred in places. The disc is of a dead-white color. It may show a network of newly formed blood-vessels. The surface of the disc is flat, the physiologic cup being filled. The vessels are diminished in calibre and white streaks accompany them. The fundus has lost some of its brightness.

THE RETINAL TYPE OF OPTIC-NERVE ATROPHY.—Optic-nerve atrophy, which is secondary to retinal degeneration, will show a picture different from those described above. The disc will appear yellowish, or, as Frost says, waxy or of the color of dirty parchment. The surface of the disc will be flat, and often it will be surrounded by a narrow ring of atrophic chorioid.

EMBOLIC ATROPHY.—Here (Fig. 2, Plate XVI) the disc shows a dense, opaque white or a yellowish-white appearance. Retinal changes are present.

Etiology.—Any cause which may produce optic neuritis may also lead to optic-nerve atrophy. Brain-tumors, injuries to the nerve-trunk, congenital malformations of the skull, erysipelas, orbital cellulitis, hemorrhage into the orbit, blows upon the head, spinal injuries, syphilis, tabes, tumors of the pituitary body, disseminated sclerosis, progressive paralysis, cerebral softening, severe hemorrhages, lightning-stroke, internal hydrocephalus, etc., are among the etiologic factors. The abuse of alcohol and tobacco is responsible for some cases. Others are congenital or possibly may be due to injury during instrumental delivery. In probably 60 per cent. of the cases there is not a discoverable cause. Many of these must be attributed to the influence of certain toxic substances which are generated in the system as the result of perverted metabolism. This view of the endogenous origin of certain cases of optic-nerve atrophy was suggested by Horner. Recent studies of the excretions of patients suffering with tobacco-amblyopia have shown excessive excretion of enterogenous decomposition-products in the urine, together with a more or less marked urobilinuria (de Schweinitz and Edsall).

Of 117 cases of optic-nerve atrophy, Derby found no cause in 71;

abuse of alcohol and tobacco, 11; syphilis, 8; injuries to the head, 9; brain disease, 6; apoplexy, 1; epilepsy, 1; locomotor ataxia, 1; meningitis, 2; optic neuritis, 2; erysipelas following lacrimal abscess, 1; mumps, 2; tumor of pituitary body, 1; following pregnancy, 1.

Pathology.—The pathologic changes will depend upon the etiologic factor. In the atrophy found in tabetic subjects the nerve-fibres lose their medullary coverings and are changed into fine fibrillæ. Fatty granular cells are found between the fibrillæ. There are no signs of a true inflammatory process. Holden believes that the primary change is an atrophy of the retinal ganglion-cells.

In the post-neuritic type of atrophy there is a formation of connective tissue in the nerve-stalk and in the nerve-head. The coats of the vessels are thickened. The sheaths of the nerve-fibres show varicosities and either

Fig. 336.—Deformity of the skull producing exophthalmos, divergent strabismus, and atrophy of the optic nerve. (RISLEY.)

shrink or disappear. In advanced cases the nerve forms a hard, distorted cord (Fig. 337).

Prognosis.—This is always grave, and will depend on the cause of the disease, the stage at which the patient applies for treatment, etc. In primary or progressive atrophy the course of the disease is slow. Several years may elapse before the patient becomes blind. The damage done to vision in the neuritic or secondary form will depend upon the amount of contraction which ensues in the inflamed tissue. A progressive narrowing of the field is a sign of grave import.

Treatment must be directed in accordance with the cause. Syphilitic cases will require mercury or potassium iodid, or both remedies. Strychnin must be given by the mouth or hypodermically. It should be administered in rapidly increasing doses and should be pushed to the limits of tolerance. If administered hypodermically the beginning dose will be $^{1}/_{100}$ grain. Given by the mouth the begining dose will be $^{1}/_{60}$ grain three times a day, increased rapidly until the patient takes $^{1}/_{6}$ or $^{1}/_{5}$ grain at a dose. Accord-

ing to Derby, improvement follows the strychnin treatment in 30 per cent. of the cases. Nitroglycerin or nitrite of amyl may be used. Other proposed remedies are preparations of arsenic, silver, iron, phosphorus, lactate of zinc, hypodermics of antipyrin, and galvanism. Vibratory massage may be tried.

If no improvement follows the use of such remedies as have been enumerated, the author removes the superior cervical ganglion of the sympathetic nerve, in the hope that an increased blood-supply will bring about improvement in vision. Although it is yet too soon to pass final judgment on this operation, the author has had one successful result out of four advanced cases of optic-nerve atrophy which he has treated by operation. To be of value, the operation should be done while the patient retains vision

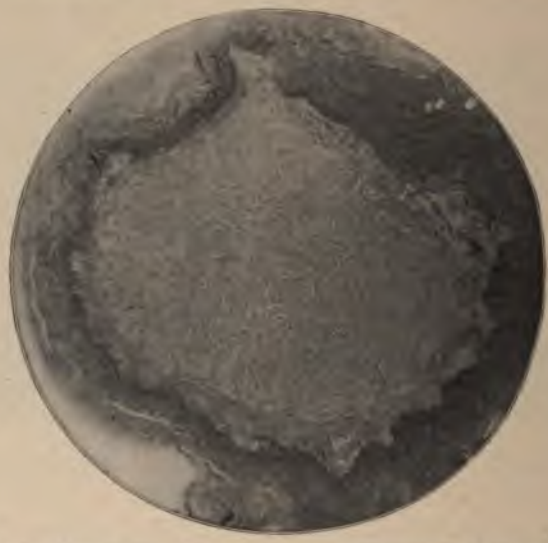


Fig. 337.—Atrophy of the optic nerve following phthisis bulbi. (Author.)

(Photomicrograph by Dr. H. P. Wells.)

equal to or better than the counting of fingers at two or three feet. Suker and Renaud have also reported cases in which sympatheticectomy was followed by improvement in vision.

Hereditary Optic-Nerve Atrophy.—This is a variety of atrophy of the optic nerve which, as a rule, affects several members of a given family at about the age of twenty years. It has been known to affect as many as six generations (Gould). The disease is found chiefly in the male members, the inheritance being through the unaffected females. About 14 per cent. of the cases reported have occurred in females. There are on record 317 cases, occurring in 80 families.

Symptoms.—The onset of the disease is sudden. It begins as an acute retrobulbar neuritis, which reaches its height in five or six weeks. After

this period it either progresses slowly or remains stationary. Central scotoma is present, which at first affects colors only and ultimately involves the form-field. Peripheral vision is either normal or slightly altered, thus permitting the patient to get about, while central vision is often so much reduced that reading is impossible. The color-changes are central at first, the periphery becoming involved later. Blue and yellow are seen more clearly than red. In anomalous cases a ring-scotoma or a sector-shaped defect may be found. Nyctalopia is a common symptom. During the stage of inflammation the patient may complain of red vision or of seeing colored stars or flames. Photophobia and headache may be present. The ophthalmoscopic signs include hyperemia of the nerve-head with slight haziness of its outline. The arteries are either normal or dilated (Leber). A fine striated appearance of the retina around the nerve-head may be present. In the later stage the papilla becomes white, with slightly hazy borders and an accumulation of pigment. The pallor of the disc generally begins in the temporal half. Atrophy always follows the neuritis.

Etiology.—Heredity is an undoubted factor. Some cases have been attributed to the abuse of tobacco and alcohol. The tower-shaped skull has been regarded by some writers as a cause, it being supposed that the optic nerve is compressed by the bone.

Prognosis.—This is unfavorable. Leber has seen a family in which all the patients recovered their vision. Partial recovery has been noted by several observers.

Treatment.—This does not differ from that which is appropriate for other types of retrobulbar optic neuritis.

Hemorrhage into the Sheath (Apoplexy) of the Optic Nerve.—Although hemorrhage into the subdural and subarachnoidal spaces of the optic nerve has been described by Magnus, de Wecker, and others, there is no evidence to show that it exists as an idiopathic affection. There are no reasons for believing that hemorrhages situated at the border of the optic disc, or in the vitreous humor, are indicative of apoplexy of the optic-nerve sheath (Gonin). Intervaginal hematoma may occur after cerebral apoplexy or following trauma. In such a case there will be rapid and great loss of vision. Early ophthalmoscopic examination may show nothing abnormal, or may give a picture resembling that which is found in embolism of the central retinal artery. At a later date the disc will be atrophic.

DISTURBANCES OF VISION WITHOUT APPARENT LESION.

Under this heading will be considered certain ocular affections (amblyopia, amaurosis, color-blindness) in which, as a rule, no changes are visible within the eye. Probably future investigations, aided by instruments and methods of greater precision than those now existing, will remove certain of the amblyopic affections from this category and place them in the list of organic diseases. The term *amaurosis* indicates com-

plete blindness, and *amblyopia* partial blindness, without discoverable fundus lesions. The term amblyopia is not applied to those conditions in which the reduction in visual acuity is due to an error of refraction, and becomes normal or approximately normal after its correction.

Congenital Amblyopia is the name applied to that condition in which there is congenital deficiency of vision and all other causes can be excluded. Generally only one eye is involved. It may be hypermetropic, myopic, and astigmatic. It may show the presence of such abnormalities as coloboma, etc., or these defects may not be present. Correction of the error of refraction does not bring the acuity of vision to the normal or near the normal grade. In such cases it is assumed that structural changes exist in some part of the ocular nervous system exterior to the eyeball. In some cases of congenital amblyopia the correction of the refraction-error is not followed immediately by improvement, but, after the use of glasses for a variable period, there comes an increase in visual acuity. Under the name *astigmatic amblyopia* Martin has described a type of congenital deficiency in vision which he believes is due to imperfect development of the finer parts of the retina, owing to asymmetrical stimulation. If both eyes are congenitally amblyopic, nystagmus will result.

Acute Amaurosis following Infantile Convulsions has been described by Nettleship, Gay, Ashby, and Stephenson. Post-eclamptic amaurosis is attributed to anesthesia of the visual centres. Aphasia and hemiplegia accompanying the visual disturbance may be transient or permanent. The amaurosis is generally transient, most patients recovering their vision in a few days. The convulsions are severe and are accompanied by coma. There are no discoverable lesions of the fundus.

Congenital Amblyopia for Colors (Color-blindness; Dichromasia; Daltonism).—Color-blindness is a subject of which much has been written and but little is known. The condition exists in about 4 per cent. of males, according to Tscherning, but is much less common (0.02 per cent.) in females. While it is usually congenital, in rare instances color-blindness is acquired.

Persons who are color-blind will (in using the worsted-skein test) pick out gray, light pink, or yellow worsteds and will add them to the green skeins. In the next test the rose-purple skein should be matched. If the patient adds blue or violet skeins, he is *completely red-blind;* if he adds the green or gray skeins, which shade toward the blue, he is *completely green-blind.* Next, he is to match red. If in addition to the red he chooses the green and brownish skeins, which are darker than red, he is *red-blind.* If he selects skeins which are lighter than red appears to the normal eye, he is *green-blind.* Various tests for color-blindness have been described in Chapter IV (page 129). Color-blindness is incurable.

Theories of Color-perception.— Of the many theories which have been advanced to explain color-perception only those of Young, Helmholtz, Hering, and Preyer will be mentioned, and these but briefly.

Young's Theory is thus set forth in his own words: "It is certain that we can produce a perfect sensation of yellow and blue by a mixture of green and red light, and of green and violet light. There are reasons for supposing that these sensations are always composed of a combination of separate sensations. This supposition at least simplifies the theory of colors; we may therefore accept it with advantage until such time as we shall find it incompatible with some phenomenon. We shall proceed, therefore, to consider white light as composed of a mixture of three colors only—red, green, and violet." This theory assumes that each nerve-fibre of the retina is composed of three secondary fibres, each one of which on irritation gives rise to the sensation of red, green, or violet. Color-blindness is explained by Young's hypothesis by the assumption that one of the secondary fibres is absent. This theory, while attractive by its simplicity, does not accord with modern observations on color-vision. It requires that from a mixture of three fundamental colors all existing hues shall be produced—an impossible condition.

The Helmholtz-Young Theory.—Helmholtz modified the theory of Young, and assumed that each spectral color irritates three fibres at once, but in different degree. He explained color-blindness by absence of one of the fibres, as Young had done. Many objections have been raised against this theory.

Hering's Theory assumes the existence of a "visual substance" which is a mixture of three others. One of these determines white and black; another, red and green; and the third, yellow and blue. It is an unprofitable task to follow this theory, which, as well as that of Helmholtz, has given rise to a vast amount of controversial literature.

Preyer's Theory assumes that the sense of color has been developed from the sense of temperature.

Amblyopia ex Anopsia (Amblyopia from Non-use; Argamblyopia).—These terms indicate a loss of vision by reason of the total or partial exclusion of one eye from the visual act. The inability to act with the fellow-eye may arise from an opacity in one of the dioptric media (leucoma of the cornea, congenital cataract, impervious persisting pupillary membrane), or it may be the result of strabismus. The amblyopia accompanying strabismus may be congenital, the defective vision causing the strabismus. On the other hand, it is well known that squinting eyes, which possessed good vision early in life, after long-continued non-use may lose even the power of fixation. Gould states that, in some cases classed with congenital amblyopia, there are fine chorioidoretinal changes in the macular region, which are the result of long-continued ametropia. Amblyopia may come from the non-use attending persistent blepharospasm (see page 189).

Treatment of this form of amblyopia consists in the early removal of the obstacle to vision, the correction of errors of refraction, and the exercising of the affected eye. The discission of cataract at a very early age is important.

Uremic Amaurosis.—Blindness without demonstrable changes in the retina or optic nerve is a symptom in persons with chronic nephritis. It is more frequently found in the acute nephritis of scarlatina, variola, and measles and in the nephritis of pregnancy. It always involves both eyes and appears suddenly, the patient becoming suddenly blind and remaining in this condition for from eight to twenty-four hours. In most cases vision is completely lost, but in some instances perception of light exists during the attack.

Severe cerebral symptoms—such as headache, vomiting, epileptiform convulsions, and coma—are rarely absent. They may precede the blindness or they may follow it. During the attack the urine is generally lessened in amount; rarely diuresis has been observed. Some authors have recorded the absence of albumin from the urine during the attack, although it was to be found both before and after the seizure. Uremic amaurosis may be the first sign of renal disease and may be associated with deafness.

Although in the majority of cases of uremic amaurosis blindness of both eyes appears simultaneously, Leber has recorded a case in which first one eye became blind, and the next day vision was lost in the other.

During the attack the pupils are either widely dilated and immobile or they react to light in spite of the complete loss of vision. Schmidt-Rimpler considers the latter condition to be the rule, to which there are exceptions. The presence of the pupillary reaction is a favorable symptom, since it indicates that the optic nerve is normal and atrophy need not be feared.

Ophthalmoscopic examination generally shows no changes in the fundus, but exceptionally transient optic neuritis, dilation of the retinal veins, and slight retinal edema have been observed.

The prognosis of uremic amaurosis is favorable. The blindness rarely fails to disappear in from twelve to twenty-four hours, and full visual acuity is reached in two or three days. During recovery transient defects in the visual field are often observed. Repeated attacks may occur at intervals of weeks or months or during repeated pregnancies. Permanent blindness from uremic amaurosis has not been observed. Where vision is permanently lost, changes will be found in the retina or in the optic nerve.

The treatment must be directed to the general condition of the patient.

Diabetic Amaurosis (Glycosuric Amblyopia).—Diabetic patients not infrequently become partly blind without showing fundus changes. Lagrange estimates the frequency of this form of visual disturbance at 4 per cent., while Schmidt-Rimpler states that it is present in 10 per cent. of cases. Mauthner has contended that diabetic amblyopia depends upon the abuse of tobacco and alcohol; but this view has been shown to be incorrect, since amblyopia has been found in diabetics who did not use tobacco or alcohol. Leber believes that the existence of diabetes renders the patient more susceptible to the toxic effect of tobacco.

The diagnosis of diabetic amaurosis will depend upon the result of an examination of the visual field and the exclusion of the use of those substances which produce intoxication-amblyopia (alcohol, tobacco, etc.). In addition to the loss of visual acuity, there is at the fixation-point and its vicinity a scotoma for red and green. Early in the case, and also later, when the patient has almost recovered, the test-object will appear dimmer than under normal conditions, without entirely vanishing. In advanced cases the scotoma becomes absolute. The peripheral part of the visual field is generally normal. The central scotoma is often of small extent. As a rule, it extends about equally in all directions from the fixation-point, but is somewhat broader on the temporal side. Generally it does not assume the shape of an oval, involving both the fixation-point and the blind spot, which is particularly characteristic of tobacco- and alcohol- amblyopia (Groenouw). Since the scotoma may be easily overlooked, small test-objects should be used in the examination.

Anomalous cases occur in which the field is concentrically contracted or hemianopic. While most cases of diabetic amblyopia will show the form of central scotoma which has been described, there are others in which careful examination fails to show the presence of a scotoma, contraction of the field, or of retinal changes (Cohn, Schmidt-Rimpler). Hemianopsia may be present alone or with other cerebral symptoms. The pathologic change which is responsible for this condition is undoubtedly a lesion of the papillo-macular bundle of optic-nerve fibres. Microscopic examinations by Edmunds and Nettleship, Schmidt-Rimpler, Grosz, and others have confirmed this opinion.

The prognosis of diabetic amaurosis concerns the continuance of the condition and the life of the patient. In general terms it may be said that the prognosis is not unfavorable. Often a proper regulation of the diet and appropriate internal treatment will result in restoration of vision; but the prognosis is less favorable if there is great reduction of visual acuity and a large central scotoma. Hirschberg and Schirmer regard the prognosis as unfavorable, having seen such patients die within a year.

The treatment will be that which is appropriate for the general condition of the patient, and is explained in works on the practice of medicine.

If a diabetic patient is addicted to tobacco or alcohol, their use should be discontinued and full doses of strychnin should be given.

Amblyopia from Hemorrhage.—Temporary amblyopia or permanent loss of vision may follow a severe hemorrhage from any one of several organs. Fries collected 106 cases of visual disturbance following hemorrhage (36 per cent. from the stomach; 25 per cent. from the uterus; 7 per cent. from the nose; 5 per cent. from accidental wounds; 25 per cent. from intentional loss of blood by venesection, leeching, or cupping). The affection, which is found more frequently after than before the fortieth year, is seen rarely in previously healthy individuals who are accidentally wounded (*e.g.*, soldiers in battle). As a rule, both eyes are in-

volved in about the same degree; in 10 to 15 per cent. only one eye is affected. When the condition is bilateral, it is rarely the case that one eye is entirely blind and the other only slightly affected. According to Pergens, blindness following hemorrhage from the stomach is twice as frequent in men as in women. The age of the youngest recorded case of amblyopia from hemorrhage was two years; the oldest patient was seventy-seven years of age.

SYMPTOMS.—The loss of vision may appear during or immediately after the hemorrhage or some days or weeks later. The patient may suffer a severe hemorrhage without visual disturbance, only to have the vision much reduced or entirely lost after a recurrence of the bleeding. In repeated hemorrhages vision may become less with each attack, while some improvement or entire restoration is noticed during the intervals. In 25 per cent. of all cases the loss of vision comes during or immediately after the hemorrhage (Groenouw). In 20 per cent. it appears within the first twelve hours. If delayed, the loss is noticed generally between the third and the sixth days, but may not appear until a period of two or three weeks has elapsed. In cases of severe hemorrhage, leading to unconsciousness, the patient on awakening will notice the blindness or may think that he is in the dark: *i.e.*, that it is night-time. Cerebral symptoms—such as headache, syncope, exhaustion, cardiac palpitation, and severe pain in the back of the neck and about the base of the skull—are often present.

The external appearance of the eye is nearly normal. In complete blindness the pupils are dilated and do not react to light. If the blindness is unilateral, the pupil of the affected eye will react synergistic with that of the normal eye.

The ophthalmoscopic appearance will vary with the time of examination. In the few cases which have been examined immediately after the advent of blindness the papilla has presented washed borders, slight swelling, with a surrounding area of slight opacity, which extends into the retina as far as the macular region and gradually diminishes peripherally. Small, brilliant spots also have been seen in the retina. In the majority of cases the ophthalmoscopic condition is either normal or negative. Ulrich in some cases saw the papilla pale, the arteries small, and the veins somewhat full. On the papilla itself the veins were narrow and of a bright-red color. Retinal hemorrhages were present.

In unfavorable cases, in which the result is atrophy of the optic nerve, the papilla appears white; the arteries are narrow. This is the usual condition in patients who were first examined long after the advent of amblyopia. The changes in the eyeground and the nerve-atrophy due thereto may be partial. Often good visual acuity will exist in spite of the presence of a white color of the optic nerve. At all events, the result of the ophthalmoscopic examination does not correspond with the degree of loss of vision; in spite of pronounced fundus changes, good visual acuity may be present, and *vice versa*.

The visual field generally shows contraction, yet its form is scarcely characteristic; there is slight concentric narrowing. Often pronounced hemianopic forms are present, or a quadrant is wanting in the upper or lower half of the field. Uhthoff saw a case in which only the nasal half of the field was preserved, the color-fields for blue and red were much contracted, and in the blind temporal half two small islands were functionating. Central scotoma with normal peripheral field was noticed by Mandelstamm and Schmidt-Rimpler. The defect may exist only for colors or an absolute central scotoma may be permanently present. In the cured or improved cases there are generally defects in the visual field; rarely does the field become entirely normal.

The light-sense has been but little examined. Pergens found it completely abolished. The central and peripheral color-sense is often materially lost, yet it may be normal or there may be narrowing of the color-fields.

Retrobulbar neuritis with central scotoma was seen by Uhthoff in two women. Borsch observed it in a man with severe gastric hemorrhage.

PATHOGENESIS.—A few of the hypotheses which have been advanced to account for the condition are: hemorrhage into the optic-nerve sheath (von Graefe, Leber); stasis in the retinal veins (Ulrich); thrombosis of the central retinal artery (Theobald); inflammation of the optic nerve with subsequent atrophy (Hortsmann and Hirschberg). The blindness which immediately follows hemorrhage is due to anemia of the brain (Fries). According to Samelsohn, complete amaurosis with retention of the pupillary reflex is caused by a lesion, probably an edema, of the visual centres. It is probable that the pathologic change may be either cerebral, neural, or retinal.

PATHOLOGY.—Few examinations have been made of the eye and its adnexa after loss of vision from hemorrhage. Hirschberg found atrophy of the optic nerves, in a patient who had lost the vision of the right eye and retained vision of $^1/_2$ in the left, after hematemesis occurring three and one-half years previously. The atrophy was complete in the right nerve and partial in the left. Ziegler found fatty degeneration of the optic nerve and retina. The examination was made twenty-three days after blindness from hematemesis. In the case of a woman aged 23 years, who became blind on the second day after severe hemorrhage during parturition, and died from edema of the brain eight weeks later, Raehlmann found the lumen of the retinal arteries much diminished from endarteritis fibrosa. A similar change was present in the veins. Holden's experiments on dogs and rabbits showed degeneration of the ganglion-cells of the retina, including their long processes, which form the centripetal fibres of the optic nerve.

PROGNOSIS.—The duration of the amblyopia varies from a few minutes to several hours. In most patients the improvement, if it occurs, appears after several days, weeks, or months. In almost 50 per cent. of cases

there is not any improvement in vision; in 33 per cent. there is some restoration; and in 20 per cent. there is complete recovery of central, without entire restoration of peripheral, vision (Groenouw). Pergens, who has reported the visual result in 50 cases of hemorrhage from the stomach, states that the result was death in 6 per cent., blindness of both eyes in 36 per cent., blindness of one eye in 18 per cent., great reduction of visual acuity in 14 per cent., and cure in 8 per cent. Recovery is possible even after perception of light has been abolished for several days, provided the pupillary reaction to light is retained. The date of the appearance of the blindness has no bearing on the prognosis. There seems to be no relationship between the amount of blood lost and the degree of improvement of vision.

TREATMENT seems to be of but very little value. A nutritious diet, rest, and the use of iron and strychnin are the most approved measures. Paracentesis of the anterior chamber and iridectomy are not to be commended.

Simulated Amblyopia (Pretended Amblyopia; Malingering).—Plaintiffs seeking damages, men desiring to avoid military duty, and other persons may claim the existence of blindness of one or of both eyes. An existing defect may be exaggerated. Atropin may be used secretly to assist in carrying out the deception. Intentional injuries of the eyes may be inflicted for the purpose of exciting sympathy or to avoid conscription. Generally the malingerer pretends that only one eye is involved. The tests which are designed for the exposure of such a case are described in Chapter IV (page 151). If blindness of both eyes is simulated only careful observation of the patient over a prolonged period will expose the deception. An attempt should be made to watch the patient secretly.

Nyctalopia (Functional Night-blindness).—In this affection the sensibility of the retina to light is diminished. It occurs suddenly in debilitated subjects after exposure to bright sunlight. Central vision is obscured, the patient seeing as through a cloud. According to Krienes, the accompanying symptoms are contraction of the field for white, reduction in central quantitative color-vision, dread of light, undue dilation of the pupils under reduced illumination, paresis of accommodation, erythropsia, xanthopsia, and epithelial xerosis.

"Nyctalopia has been observed endemically among malarial subjects, scorbutic sailors, and among children in the public schools" (Snell). It has been found among Russians who have fasted during Lent, and among persons who have been deprived of proper food. It occurs chiefly in the springtime. Sleeping in the moonlight is said to be a cause. Nyctalopic patients see poorly on dark days and well on bright ones. The affection accompanies xerosis, and is found in some retinal and chorioidal diseases.

PROGNOSIS is favorable. The affection often disappears after several weeks or months. It tends, however, to return in the spring or summer of the following year.

TREATMENT.—It has long been the belief of the laity that the ingestion of the liver of the sheep will cure night-blindness. This opinion has been confirmed by scientific observers. The patient should ingest from 6 to 8 ounces of the liver of the goat, sheep, or ox, three times a day. The liver is to be fried in oil and is seasoned with spices. Buchanan, who treated twenty cases in this manner, states that five or six days are sufficient to effect a cure. Under ordinary tonic treatment such cases are not improved for several months. The liver treatment should be followed by a course of codliver-oil and ferruginous tonics. The night-blindness of retinitis pigmentosa is not improved by this treatment.

Hemeralopia (Day-blindness).—Hemeralopic patients see better on dark than on bright days. The same state of affairs is true of patients suffering with toxic amblyopia. Hemeralopia occurs in persons who have been long removed from light, and in those who have congenital defects, such as albinism, irideremia, coloboma of the chorioid, coloboma of the iris, etc. It is said to be congenital and to be associated with congenital amblyopia. Hemeralopia may be caused by exposure to bright light, such as reflection from snow and ice. Eyestrain from errors of refraction is said to be a cause. Photophobia and dazzling sensations are annoying symptoms.

TREATMENT will include the correction of errors of refraction or of muscle-balance, the use of smoked glasses, and the administration of tonics. The patient should be instructed gradually to become accustomed to bright light.

Scotoma Scintillans (Amaurosis Partialis Fugax; Teichopsia; Flimmerskotom).—These terms are applied to an amblyopia of central origin. The patient while suffering with vertigo notices before the eyes a bright light, which rapidly increases until finally he can scarcely see. Some patients describe the first appearance as that of a small spot situated near the fixation-point. Within the area of this spot objects become invisible. The scintillating area spreads rapidly. It seems to be formed with flickering points arranged in a zigzag manner. The amaurosis disappears in from fifteen to thirty minutes, and is succeeded by nausea, vomiting, and headache (migraine). The affection is bilateral, and often involves but one-half of the visual field (the homonymous half).

Scintillating scotoma is supposed to depend on circulatory disturbances in the occipital lobes. When occurring at long intervals it has no significance; if often present it may indicate impending cerebral lesions. Thus it may precede hemianesthesia, hemiplegia, brain tumor, apoplexy, and bulbar lesions. In some cases it is due to cerebral anemia. Swanzy states that fatigue, long reading, and hunger may cause it. It is not infrequent among physicians and other brain-workers.

TREATMENT will include a cessation of brain-work, the use of alcoholics in moderation, the securing of sufficient sleep, and the taking of appropriate exercise. Quinin, bromid of potassium, caffein, and strychnin have

been used. Neustätter advises the administration of validol in 20-drop doses.

Snow-blindness.—Exposure to blinding reflections of the sun produces a variety of changes in the conjunctiva (hyperemia and edema), dendriform ulceration of the cornea, contraction of the iris, and alterations in the retina (see page 514). Photophobia, blepharospasm, and lacrimation are early and annoying symptoms. Temporary or even permanent amblyopia may accompany the disease. Generally the symptoms disappear rapidly, the affection lasting only a few days. A scotoma may be present, with or without peripheric limitation of the field of vision. Reflection from snow may cause night-blindness. In the absence of sunlight erythema of the conjunctiva may be produced by the impact of flying particles of ice or of snow. Exposure to the x-ray and to electric flashes may cause similar symptoms. The treatment of snow-blindness includes removal of the cause, protection of the eyes by means of smoked glasses, the use of atropin and hot applications, and the instillation of a weak solution of argyrol.

Hemianopsia (Half-sight) is discussed in Chapter XXII.

Reflex Amblyopia.—This term has been applied to cases in which loss of vision is supposed to be due to reflex irritation. The condition has been attributed to intestinal parasites, to decayed teeth, and to diseases of the naso-pharynx, uterus, or ovaries. If caused by carious teeth the amblyopia will be unilateral or, if bilateral, will be greater on the side of the dental disease. Vision may be slightly reduced or may be limited to perception of light. Removal of the source of irritation is followed by recovery of visual acuity.

Hysteric Amblyopia occurs in both sexes, but is more frequent in neurotic females. It is usually associated with other signs of hysteria. As a rule, but one eye is affected and the loss of vision is complete. Usually the pupil reacts to light. In some cases the pupillary reaction is abolished temporarily—*i.e.*, the pupil is dilated and immobile. In cases of less severity there may be a central scotoma and partial color-blindness. Perimetric examination of such subjects will often show the field for red and green to be larger than that for white. Complete reversal of the color-fields has been noted. Ophthalmoscopic examination shows nothing abnormal. Concomitant hysteric symptoms are hemianesthesia, loss of various (pharyngeal, corneal) reflexes, blepharospasm, ptosis, monocular diplopia, micropsia and megalopsia, conjugate deviation of the eyes, and the symptom-complex called "retinal asthenopia."

Diagnosis may be difficult. Symptoms which are attributed to hysteric amblyopia may long precede the advent of serious cerebral disease (Friedenwald).

Prognosis is ultimately favorable, although the amblyopia may persist for weeks or months.

Treatment will include suggestion, tonics, rest, massage, electricity, etc.

Colored Vision.—Red vision, *erythropsia,* occurs not infrequently after cataract extraction, particularly in patients who neglect to wear smoked glasses for several weeks after the operation. Erythropsia has also been observed in persons who have had no operation or inflammation of the eyes. In such cases it is attributed to exposure to intense light or to irritation of the cortical visual centres. The use of coffee may cause red vision. It sometimes follows fasting and fatigue due to excessive near work. It is not uncommon among neurotic school-children. Blue vision, *cyanopsia,* occasionally appears after cataract extraction, and is attributed to the diffusion of light caused by thin layers of cortical substance which remain in the pupillary area (Becker). With the absorption of the cortical masses the blue vision disappears. It also occurs in overworked, nervous subjects. Yellow vision, *xanthopsia,* is a symptom occurring in santonin poisoning, and is noticed by patients who are suffering with glaucoma and optic-nerve atrophy. *Green vision* has been noted by Dodd, who collected 13 cases. One of these followed cataract extraction, 1 was sequent to a wound, and 8 suffered with diseases of the optic nerve, retina, and chorioid. In 2 cases the green vision was noticed only when the patients were indoors. In glaucoma the patient may complain of variously colored halos around lamplights. Persons with blind eyes tell of phosphene-experiences which are probably due to an irritation of the visual centres.

TREATMENT.—Bromids, iodids, pulsatilla, and gelsemium have been recommended.

Malarial Amblyopia.—Transient amblyopia may occur in persons who are suffering with malaria. The pupils are widely dilated and vision is lost. Fundus changes are absent and vision returns after the administration of quinin and other appropriate remedies.

Micropsia, Megalopsia, and Metamorphopsia.—Objects may appear too small, *micropsia;* too large, *megalopsia;* or distorted, *metamorphopsia.*

These symptoms may be complained of in hysteria, or may exist in any condition which causes a separation or an approximation of the retinal elements (rods and cones). Syphilitic chorioidoretinitis is a frequent cause of micropsia and megalopsia. After the correction of a refraction-error of high degree the patient may complain that objects are too small, too large, or are distorted.

Benson has recorded a case of monocular micropsia, which was attributed to weakness of the accommodation from an attack of diphtheria. The micropsia and megalopsia of hysteria are attributed by Parinaud to spasm of the accommodation.

Dudley has reported the cases of three middle-aged men, each of whom complained of (right) monocular *metamorphopsia varians: i.e.,* the distortion of the object viewed was constantly changing (see Fig. 338). Since refraction and retinal changes did not exist, he concluded that the cause must be mental. Savage believes that the condition depends on retinal changes which are too minute to be visible ophthalmoscopically.

CHAPTER XVII.

GLAUCOMA.

NORMAL INTRA-OCULAR TENSION.

WITHIN the eye are three kinds of fluid: blood from the blood-vessels; lymph, which is present in the lymph-spaces of the uveal tract and perivascular lymph-channels, its amount depending on the blood-pressure; and the intra-ocular fluid, which is a secretion of the epithelium covering the ciliary body and is found in the aqueous and vitreous chambers. The intra-ocular fluids obtain exit in various ways. The major portion escapes via the anterior chamber, the spaces of Fontana, the canal of Schlemm, and thence into the anterior ciliary veins (Leber). A portion escapes via openings on the anterior surface of the iris and thence into the iridal veins by filtration (Nuel). A small part passes into the vitreous humor and escapes via the lymph-spaces which surround the central retinal vessels. The normal intra-ocular fluid (aqueous and vitreous humors) is composed of 99 per cent. water, 1 per cent. salts and extractives, with a trace of albumin. Filtration of such fluid can occur readily. If the albumin is increased in quantity, as occurs in glaucoma, filtration becomes more difficult.

Normal pressure within the eye is equal to a column of mercury 26 to 28 millimetres in height, varying with race, age, and altitude. Uniformity of pressure is maintained by a nervous mechanism which is not thoroughly understood. The sympathetic nerve has a potent influence upon intra-ocular tension. Stimulation of the cervical portion of this nerve causes dilation of the pupil, increased blood-pressure, increased secretion from the ciliary body, and contraction of Müller's muscular fibres. Excision of the superior cervical ganglion of the sympathetic nerve causes an opposite effect, viz.: contraction of the pupil, decreased blood-pressure, diminished secretion from the ciliary body, and relaxation of Müller's fibres. Dividing or irritating the trigeminal nerve in animals has lead to contradictory results. Donders found that division of the nerve produced lowered tension, while its irritation was followed by increased intra-ocular pressure. Wegner found that similar experiments gave negative results.

GLAUCOMA: GENERAL CONSIDERATIONS.

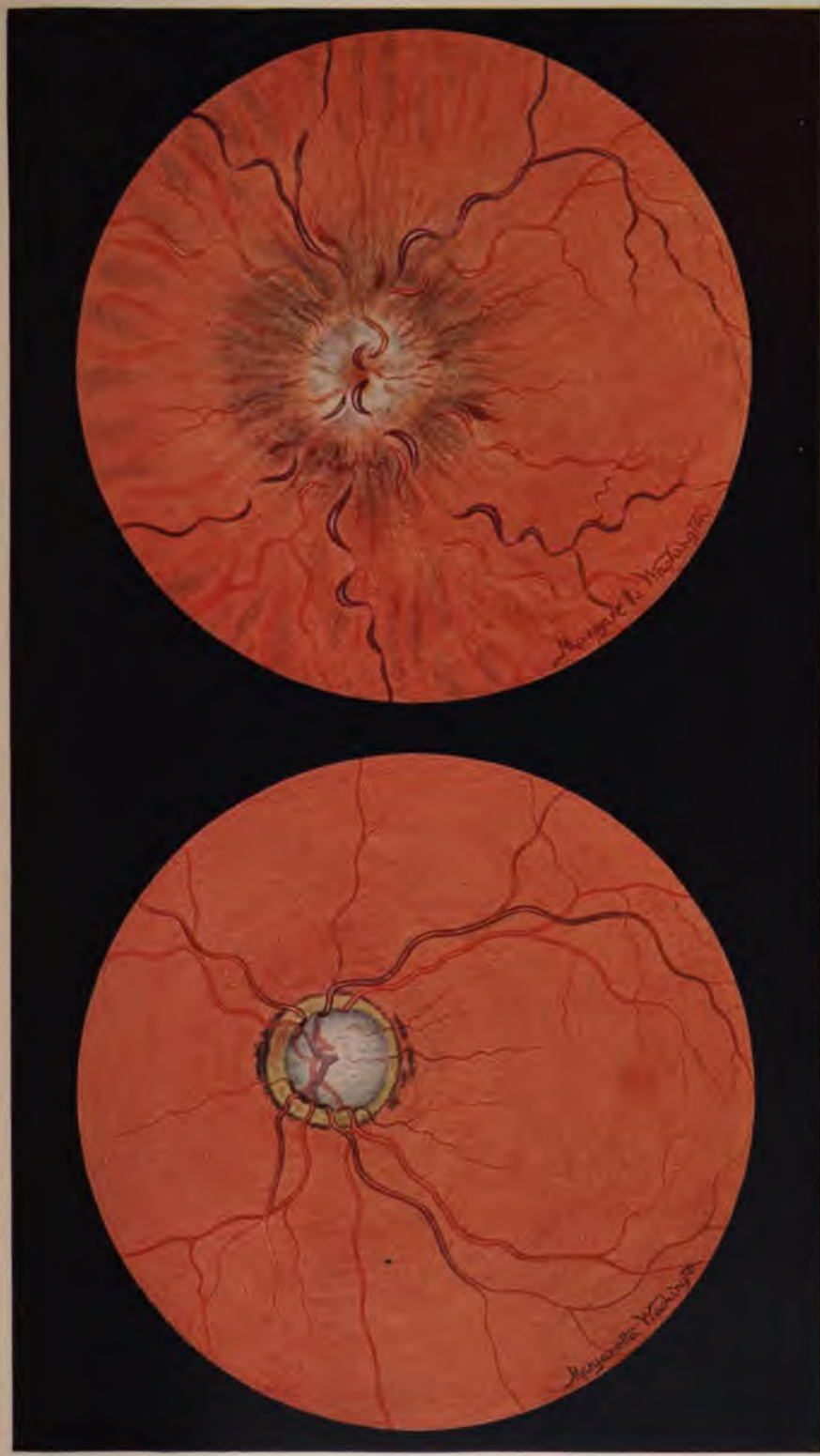
Glaucoma has been defined as increased intra-ocular pressure plus the causes and results of such pressure. This definition, which is credited to Priestley Smith, leaves much to be desired. The signs of glaucoma are well known. Its effects upon the ocular structures and functions are thoroughly understood. The limits of its therapeusis are painfully apparent,

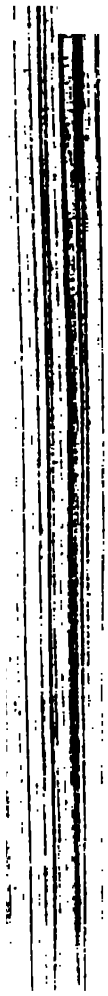
Opticociliary Neurotomy was proposed by Boucheron in 1890. Under general anesthesia, the internal rectus muscle is exposed and is secured by passing a suture through it at a point about ten millimetres behind the cornea. The muscle is then cut anterior to the suture. The optic and ciliary nerves are then cut. The muscle is sutured and a compress bandage is applied. This operation has been superseded by

Opticociliary Neurectomy.—This operation includes section of the internal rectus muscle, division of the optic and ciliary nerves close to the optic foramen, and excision of the optic nerve. In order to accomplish the excision the globe must be turned so that its posterior part becomes visible. The eyeball is replaced, the muscle is sutured, and a bandage is applied.

Value of these Procedures.—Opticociliary neurotomy and neurectomy were proposed for the purpose of preventing sympathetic ophthalmitis. They are not sure means of prophylaxis, several cases having been reported in which sympathetic inflammation occurred long after the operation had been performed. For the prevention of sympathetic ophthalmitis neurotomy ranks far below enucleation, evisceration, or neurectomy. Since regeneration of the nerves occurs, opticociliary neurectomy is not a sure means of preventing sympathetic disease.

PLATE 23





but the real cause of the disease is unknown. Except for those cases in which increased tension is due to changes in other parts of the eye,—viz.: secondary glaucoma,—the ophthalmologists of to-day have been until recently almost as much in the dark as to its etiology as were those of fifty years ago. Recent investigations have shown that under the name glaucoma are included several different diseases. Late observations indicate that changes in the sympathetic nervous system have much to do with the production of idiopathic glaucoma. The author believes that primary glaucoma should be defined as a disease of the sympathetic nervous system producing increased intra-ocular pressure.

Glaucoma may be congenital or acquired, primary or secondary to some other disease, acute or chronic, inflammatory or simple. Always, however, it is a progressive disease, and tends to destroy vision. It may begin with hemorrhages from the retinal vessels, or these may never rupture, or the glaucomatous process may last for a long period and the hemorrhages appear in the last act of the drama. The subject will be considered under the following heads: (1) glaucoma simplex, (2) chronic irritative glaucoma, (3) acute inflammatory glaucoma, (4) hemorrhagic glaucoma, and (5) secondary glaucoma. These divisions, while necessary for teaching purposes, are somewhat arbitrary, since all sorts of gradations exist. Thus, a case of glaucoma simplex, after a long period, may end in an attack of the acute inflammatory type and be followed by hemorrhages into the eye.

Etiology.—Glaucoma is chiefly a disease of advanced life. The liability of females is greater than that of males in the ratio of six to five. At the age of 65 years the liability to an attack of glaucoma is twice as great as at 45 years, and 100 times as great as at 15 years (Priestley Smith). The liability of advanced life is thought by Priestley Smith to be due to the growth of the lens. While the cornea attains its maximum diameter about the fifth year, and the globe is of full size at the twentieth year, the lens continues to grow from youth to old age. During the period between 25 and 65 years it adds one-tenth to its diameter and one-third to its volume (Priestley Smith). The lens, pressing against the ciliary processes, causes the base of the iris to encroach on the filtration angle, and thus excretion of aqueous humor is hindered. Small eyes are especially liable to primary glaucoma. An eye with a small cornea and an antero-posterior diameter 1 or 1.50 millimetres less than the normal is very likely to become glaucomatous. Heredity would seem to be a factor only when associated with congenital smallness of the globe. Certain races seem more liable to glaucoma than others. It is said that the disease is particularly frequent among Egyptians and Jews and among the negroes of Brazil. The influence of hypermetropia and accommodative strain seems undoubted. Mydriatics—such as atropin, scopolamin, cocain, homatropin, and euphthalmin—have been known to cause glaucoma in eyes previously presenting no sign of disease. When the filtration angle is already small, the folding and thickening of the iris, which are produced by the mydriatic, will cause a

further blocking, and glaucoma results. Congestion of the uveal tract is a factor. Many cases can be traced to exposure, bronchitis, cold in the head, heart disease, hepatic derangements, constipation, all of which are conditions in which the venous system is engorged. It is a common observation that a patient with glaucoma feels much better and his eye looks better after the alimentary tract has been emptied. Grief, anxiety, worry, and the loss of sleep are direct causes of primary glaucoma. Operation on one eye is often followed by an attack of glaucoma in the other eye. Slight local injury, such as an abrasion of the cornea, may cause a glaucomatous attack.

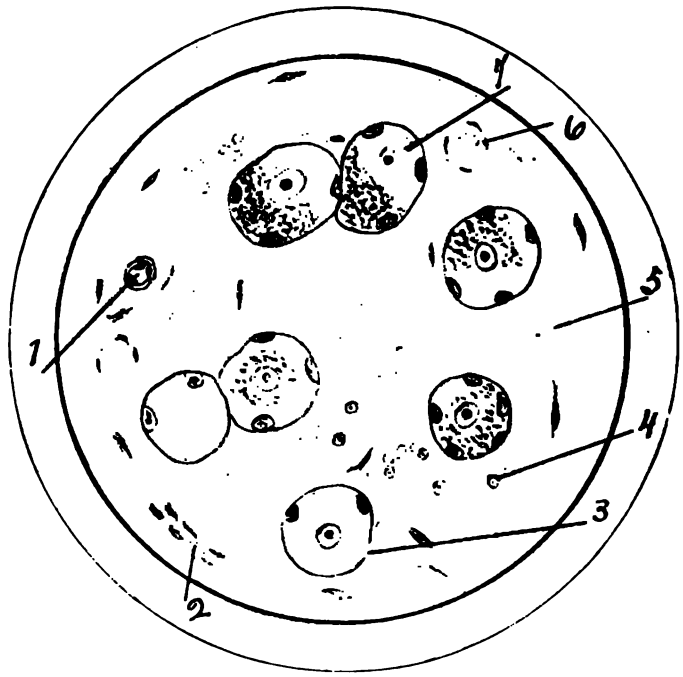


Fig. 339. Section of normal superior cervical ganglion. (Author.)

Original drawing by Dr. Carl Fisch.

1, Artery. *2*, Capillary. *3*, Nerve-process. *4*, Medullated fibres. *5*, Bundle of nerve-fibres. *6*, Bundles of non-medullated fibres cut across. *7*, Nerve-cell.

(Leitz: ocular 4, objective 6.)

Certain cases of glaucoma must be attributed to disease of the blood-vessels leading to thrombosis, hemorrhage, or exudation.

Finally, the sympathetic nervous system has much to do with the production of glaucoma. This view is suggested by the fact that, in the cases in which the author removed the superior cervical sympathetic ganglia for the cure of glaucoma, the microscope showed marked pathologic changes. Laqueur noticed that the exciting causes of mild attacks in the so-called premonitory stage of glaucoma are conditions of nervous depression (cold, hunger, fatigue, fright, anger, sleeplessness, etc.) which are associated with

dilation of the pupil, while the conditions under which these premonitory attacks disappear (warmth, food, sleep) produce contraction of the pupil.

Pathology.—The pathologic changes in glaucoma are found in the cervical portion of the sympathetic nerve, in the tissues of the eye, and in the intra-ocular contents.

Changes in the Sympathetic Nerve.—Microscopic sections of the superior cervical ganglion of the sympathetic nerve show a marked hyperplasia of connective tissue, which often results in the division of the ganglion into small groups of nervous elements separated by broad bands of fibrous elements. The walls of the blood-vessels show sclerosis. The connective-tissue sheaths of the ganglionic cells are much thickened, and an infiltration of small round cells is present in the hyperplastic tissue.

Fig. 340.—Angle of the anterior chamber of a normal eye. (Author.)
(Photomicrograph by Dr. H. P. Wells.)

The ganglionic cells are markedly pigmented. Together with a number of cells normal to all appearance there are great numbers showing different stages of degeneration. As a rule, the nucleus, besides losing part of its peculiar staining property, assumes the parietal position. The nucleus is reduced in size or even missing in a large percentage of the cells. While in some cells the chromatic elements are well preserved, in others the process of chromatorrhexis and chromatolysis can be followed through all of the stages. Only comparatively few cells are seen showing the normal dendriform processes. Often the processes are short, ending bluntly, or even disappearing altogether. The general peripheral network of processes is much reduced in volume and compressed by the pressure of the connective-tissue formation. Only very few medullated fibres are seen.

The general pathologic aspect is that of a decided sclerosis, originating in inflammatory processes going on in, and starting out from, the walls of the vascular structures. The changes of the nervous elements may not be idiopathic, but are probably due to pressure and inhibited nutrition. While these changes are suggestive of the sympathetic origin of glaucoma, it must be acknowledged that similar pathologic lesions of the sympathetic nerve exist in optic-nerve atrophy and in exophthalmic goitre.

THE MACROSCOPIC CHANGES IN THE EYEBALL are chiefly a blocking of the angle of filtration and an excavation of the head of the optic nerve. These alterations can be accounted for by the theory that pathologic changes in the sympathetic nerve cause increased intra-ocular pressure. In response

Fig. 341.—Angle of the anterior chamber of a glaucomatous eye. (AUTHOR.)

(Photomicrograph by DR. H. P. WELLS.)

to hypertonia, the lens is pushed forward, and the iris becomes adherent at its periphery to the cornea. The escape of the aqueous humor is hindered; the optic-nerve entrance, being the weakest part of the globe, is pushed backward. The escape of blood through the veins of the chorioid is interfered with, by contraction of the unstriped peribulbar muscular fibres, which are found in the capsule of Tenon. It is probable that the diseased sympathetic causes an increase in the secretion of the aqueous humor. Thus, increased secretion, on the one hand, and diminished excretion, on the other, bring about a condition which ends in the complete disorganization of all of the ocular structures. That this is a reasonable hypothesis is proven by the fact that, if the changes have not progressed too far,—*i.e.*, if the root of the iris is not too firmly fastened to the cornea,—excision of the

sympathetic nerve will cause a diminution of tension, a contraction of the pupil, and an improvement in the intra-ocular circulation.

THE ANATOMIC CHANGES IN GLAUCOMATOUS EYES are all due to increase of tension. The venæ vorticosæ being compressed easily, because of their oblique course through the sclera, the venous blood from the uveal tract passes from the eye in large part through the anterior ciliary veins, which become enlarged and tortuous. In acute inflammatory glaucoma the uveal tract is the seat of inflammatory edema. Synechiæ are rare, and exudations of magnitude are never seen. The cloudiness of the cornea is not a true inflammation, but an edema. The iris is similarly affected, as is shown by discoloration and swelling. In response to pressure the lens moves forward, decreasing the depth of the anterior chamber; and swelling of the ciliary processes results in the approximation of the iris-base to the periphery of the cornea, as shown in Fig. 341. Compression of the ciliary nerves causes loss of sensation in the cornea and dilation of the pupil, with paralysis of the iris. The pain of glaucoma comes from the compression of nerve-filaments in the ciliary body and iris. Loss of vision is due to three results of pressure: (1) the opacity of the cornea, explained above; (2) ischemia of the retina, due to pressure, contracts the visual field; and (3) pressure on the optic-nerve fibres causes atrophy. In glaucoma simplex loss of vision is due chiefly to excavation and to atrophy of the nerve.

It is now necessary to consider more explicitly the effects of increased intra-ocular tension upon the various structures of the eye and their respective functions. The *eyelids* in acute cases may show edema. The *conjunctiva* shows general hyperemia, and in acute types it becomes edematous (chemosis). It is of a dusky color. The *anterior ciliary veins,* which normally give exit to a small part of the blood from the uveal tract, in glaucoma are much enlarged by reason of the blocking of the venæ vorticosæ through which normally the major portion of the blood from the uveal tract passes from the eye. The *anterior ciliary arteries* become hypertrophic in cases in which the increase of tension is of long duration. If increase of tension occurs slowly the vascular changes are slight. The *sclera* becomes distended in the glaucoma of childhood (hydrophthalmos). In adults it is a fixed and unyielding structure. In eyes which have long been glaucomatous there is a separation of the scleral bundles. The *cornea* becomes edematous. Microscopic examination shows spaces between the epithelial and anterior elastic layers as well as between the epithelial cells. The spaces between the anterior layers of the lamellæ are distended with albuminous fluid. These changes are apparent clinically as a superficial haziness which rapidly disappears when the intra-ocular tension is lowered. Vesicles may form on the surface of the cornea. The haziness causes the patient to complain of the appearance of a halo around lights. Anesthesia of the cornea is a common symptom in glaucoma, and is attributed to compression of nerve-fibres either in the cornea or on the long ciliary nerves lying in their scleral grooves.

The *anterior chamber* in primary glaucoma is shallow from pressure in the vitreous chamber forcing the lens forward. In congenital glaucoma, as well as in some secondary cases, the iris is primarily adherent at its periphery to the posterior surface of the cornea, thus blocking the angle of filtration. In such an event the anterior chamber becomes deepened. The *iris,* in recent cases, is pressed against the posterior surface of the cornea. Thus, the iridal vessels and nerves are compressed. If the increase of intra-ocular tension comes suddenly, the iris shows edema, venous engorgement, and change in color. At a later period it undergoes atrophy. The vessels are emptied, become sclerotic, and undergo hyalin degeneration. The stroma disappears and the uveal pigment projects into the pupillary area (ectropion uveæ). In acute glaucoma the pupil is dilated, pressure causing loss of function in the sphincter muscle. If tension is relieved by an early operation, the function is restored. In old cases of glaucoma the pupil is permanently dilated. It may be round, but generally is somewhat oval. Adhesions between the iris and cornea can be readily separated in recent cases. In old ones the iris shows exudation, small-cell infiltration, and firm adhesion to the cornea. In glaucoma simplex there is often an absence of dilation of the pupil and an absence of atrophy of the iris.

The effect of increased pressure upon the *ciliary body* is shown in the apparent rapid increase of presbyopia. The patient will accept stronger convex lenses for reading than his age warrants, and will add to the strength of his glasses at short intervals. In acute types of glaucoma the ciliary processes become swollen and edematous. They press against the peripheral portion of the iris. Later they become atrophic. In glaucoma simplex these changes are slight. In old cases the ciliary muscle becomes atrophic. Pain, which is due to compression of the nerve-fibres of the ciliary plexus by the edematous ciliary processes, is frequently present in acute types of the disease and is often referred to other branches of the trifacial nerve. If the increase of tension is but slight, or if it comes on slowly, as in glaucoma simplex, pain is either absent or is insignificant. Early in the history of a case of acute glaucoma the *chorioid* is edematous from venous obstruction; later it becomes atrophic. In the simple form of the disease the atrophy is less marked and is slow to appear. Such a case will often show a "tessellated fundus." Atrophy of the chorioid around the optic disc is often seen in old cases of glaucoma. An area of sclera surrounding the disc is then exposed to view. In the equatorial region, where the venæ vorticosæ obtain exit, the chorioid is often atrophic.

The *crystalline lens* also suffers changes in glaucoma. In acute cases it is pushed forward and its refractive power is lessened. The effect is to make the emmetropic eye myopic. In old cases of glaucoma the lens becomes opaque (glaucomatous cataract), and appears of a bluish-white color with a silk-like lustre (Fig. 5, Plate XII). This form of cataract appears in eyes which are undergoing glaucomatous degeneration, and hence are blind. It is to be distinguished from that form of lenticular opacity which accidentally

occurs in an eye which has glaucoma (cataract in a glaucomatous eye). It is evident that the latter condition may offer a favorable prognosis. Thus, the surgeon, in such a case, makes an iridectomy to reduce the intra-ocular tension and some weeks later extracts the lens. Traumatic cataract also may occur in an already glaucomatous eye. The *intra-ocular* fluids are altered in glaucoma, as will be explained in a succeeding paragraph.

The *retina* suffers from increased intra-ocular tension, the first effect being an obstruction to the entrance of arterial and to the exit of venous blood. Arterial pulsation is either present or can be elicited by slight pressure made upon the globe through the intervening eyelid. Sclerosis of the vessels, with hyalin degeneration, and hemorrhages are not infrequent in glaucomatous eyes. The retina shows the effect of increased pressure by a disturbance of function or by atrophy. Loss of vision may appear suddenly in acute glaucoma or it may develop slowly in the chronic form of the disease. So long as atrophy of retinal tissue has not taken place a restoration of vision is possible. Reduction in vision begins peripherally and is manifested as a contraction of the visual field, which is generally first observed on the nasal side (temporal half of the retina). The arteries and nerve-fibres which pass to the temporal portion of the retina must run a greater distance than those which supply the nasal side. Hence the early loss in the nasal part of the visual field. The field may be concentrically contracted or irregularly lessened. Generally the field assumes an oval shape with its chief area on the outer side of the point of fixation. There is usually commensurate loss of central visual acuity, but in glaucoma simplex there may be marked contraction of the visual field, with retention of normal central vision. In some cases there is a central or paracentral scotoma. The color-fields are generally contracted commensurately with the loss of the field for white.

The *optic nerve,* which is swollen and edematous in the early stages of acute glaucoma, soon becomes excavated. The causes of the excavation are the retrocession of the lamina cribrosa and the atrophy of the nerve-fibres. The excavation involves the entire head of the nerve and may assume one of several shapes. A low grade of neuritis is often present in glaucoma, as is evidenced by the presence of round-cell infiltration in the optic nerve surrounding the excavation. The term *anterior glaucoma* is applied to those cases which show the chief alterations in the anterior ocular segment (adhesion of the iris to the cornea, closure of Schlemm's canal, etc.), while the name *posterior glaucoma* indicates that the chief changes are to be found in the optic-nerve head and its vicinity.

Changes in the Intra-ocular Contents.—Troncoso has shown that in glaucoma the composition of the aqueous humor is altered. Its density is increased. The quantity of mineral salts is greater than normal. Most striking is the increase in organic ingredients (albumin, etc.). This change in the amount of albumin present in the aqueous humor is attributed to vascular disturbance. A fluid which is loaded with albumin can be

excreted only slowly and with difficulty. The quantity of aqueous humor is less in the glaucomatous than in the normal eye (Troncoso).

Ophthalmoscopic Signs.—In glaucoma the ophthalmoscope shows a characteristic picture. The head of the nerve is excavated, the vessels are pushed toward the nasal side of the disc, and the edges of the disc often appear undercut (Fig. 2, Plate XXIII). This condition is shown by the bending of the vessels, which appear as if climbing over a ledge. The lamina cribrosa, being the weakest part of the eye, is bent backward and carries with it the vessels and nerve-fibres. The arteries become smaller than normal, the veins are engorged, and the nerve-fibres undergo atrophy. Usually the whole optic-nerve head is depressed, the excavation being complete, as shown in Fig. 343. Occasionally, in glaucoma simplex, the excavation is complete only on the temporal side of the disc, while the vessels on the nasal side are separated from the scleral ring by a narrow margin of nerve.

Fig. 342.—Head of normal optic nerve. (Author.)

Fig. 343.—Head of optic nerve in glaucoma absolutum. (Author.)

As a result of retrocession of the lamina cribrosa producing atrophy, vision—central and peripheral—is reduced and finally lost. Peripheral vision is the first to suffer, owing to ischemia. The loss is manifested by a contraction of the form-field, which is either concentric or irregular. Often the limitation of the field begins on the nasal side, the temporal part of the nerve being the first to suffer. In glaucoma simplex it is not unusual to meet with a concentric contraction of the field.

In making a diagnosis with the ophthalmoscope the observer must remember the anatomic peculiarities of different varieties of excavation: the physiologic, atrophic, and glaucomatous. A partial excavation is physiologic; a total one is pathologic. Of the latter, the excavation of atrophy is shallow, and slopes gradually. The excavation of glaucoma is shallow or deep according to the duration of increased pressure. In the shallow form the nerve-head retains a good color, while in the excavation of atrophy the papilla looks very white. If the excavation be deep and involve the whole disc, it can be due only to glaucoma. Atypical cases

are sometimes seen in which only the temporal side of the disc is excavated; the vessels are pushed to the nasal side, being separated from the scleral ring by a band of healthy nerve-tissue. In myopic eyes in which glaucoma supervenes the picture may be atypical, and repeated examinations may be necessary to establish the diagnosis.

In advanced glaucoma the whole nerve-head is deeply excavated and the papilla is of a bluish or greenish-white color. In the depths of the excavation the lamina cribrosa can be recognized by the presence of gray dots. By direct ophthalmoscopy the vessels cannot be traced from the centre of the nerve to the scleral ring, but by the indirect method they can be seen in their whole course. The direct method enables the observer approximately to measure the depth of the excavation. If the vessels, as they

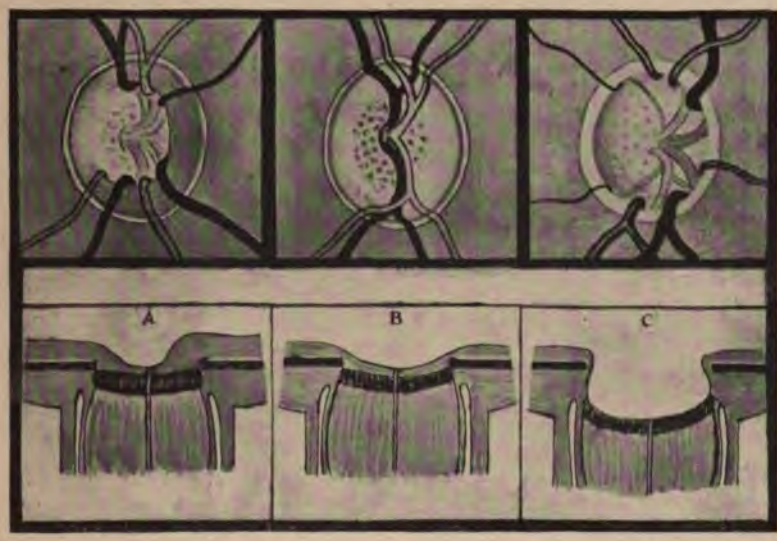


Fig. 344.—Types of excavation of the optic nerve. (JENNINGS.)

A, Physiologic excavation. *B*, Atrophic excavation. *C*, Glaucomatous excavation.

climb over the ledge, are seen distinctly without a lens in the ophthalmoscope, a concave lens will be necessary to see them distinctly at the bottom of the depression. Each dioptre represents a depth of 0.3 millimetre. Suppose, for example, the retinal vessels at the scleral ring are seen with a + 1 D. lens and the central vessels in the depressed nerve are visible clearly with a — 1 D. lens, the excavation would amount to 0.6 millimetre, the difference between the two glasses being 2 D. In using indirect ophthalmoscopy the difference in level between the scleral ring and the depressed nerve-head can be recognized only by parallactic displacement, the bottom of the excavation and the surrounding fundus seeming to move at different rates. The excavation moves more slowly than does the fundus. Thus the fundus seems to move over the disc. In old cases of glaucoma the papilla

is surrounded by a circle of atrophic chorioid—the so-called glaucomatous halo.

The loss of sight does not depend so much on the depth of the excavation as on the extent to which atrophy of the nerve-fibres has taken place. It is not unusual to find that an eye with a deep excavation possesses considerable or possibly normal central vision. On the other hand, the vision may be lost completely in a few hours—as in glaucoma fulminans—without excavation, the period being too short for excavation to occur. The color of the papilla and the size of the retinal vessels form a better guide for judgment as to visual acuity than does the depth of the excavation.

CLINICAL VARIETIES AND TREATMENT OF PRIMARY GLAUCOMA.

Glaucoma Simplex.—This disease comes so slowly and quietly that it is often overlooked. It may appear at any time between the thirteenth and ninety-sixth years, although most cases develop during middle life. Few cases have been seen in children. The refraction is usually hypermetropic, although exceptionally it may be myopic. Externally the eye may appear normal, or the sclera may be abnormally white and present a few tortuous vessels. The disease at first causes no pain, but only a feeling of heaviness in and around the eye. Central vision may be almost normal, or normal one day and foggy (V. $= {}^{20}/_{40}$ or ${}^{20}/_{30}$) the next. The field for form is somewhat contracted, and the color-field is narrowed in proportion. Tension is variable—normal at one time and slightly increased at the next examination. The anterior chamber may appear of normal depth and the pupil may be responsive to light. One eye only may be affected, in which case the corresponding pupil will be slightly larger than its fellow. Sometimes, during the period of fog, careful examination will show steaminess of the cornea. So far as the symptoms referable to the front of the eye are concerned, it is impossible to make a diagnosis. The examiner must depend on the findings of the perimeter and particularly on those of the ophthalmoscope.

For a long time in the history of a case of glaucoma simplex the media are clear and the optic nerve can be examined satisfactorily. The nerve-head may present the classic punched-out appearance, or the excavation may markedly resemble that of atrophy, except that in one part of the disc the excavation is very deep and extends to the periphery. The vessels are pushed to the nasal side, and they do not form continuous lines, but are broken in their course from the centre of the nerve to the edge of the disc. The veins may be normal or enlarged and dark. The arteries may or may not pulsate spontaneously. If they do not beat spontaneously, pulsation is readily elicited by slight pressure on the upper lid. In typical cases the diagnosis of glaucoma simplex can be made easily with the ophthalmoscope, but atypical cases may puzzle the surgeon and may require repeated examinations. A map of the visual field is of importance. Central form-

and color- vision are good, while the peripheral fields may be much contracted. The usual change of the form-field is a loss in the nasal portion; or there is concentric contraction; or sectional defects, especially in the upper field, are found; or the whole field will be contracted so that only an insignificant patch remains. In some cases scotomata are found. Usually the color-field is lost commensurate to the loss in the form-field.

Spontaneous pulsation in the retinal arteries, while highly suggestive of glaucoma, is not pathognomonic, since the retinal arterial pulse is seen also in insufficiency of the aortic valves, in exophthalmic goitre, in aneurisms of the aorta and innominate arteries, and sometimes in neurasthenia and chlorosis.

DIAGNOSIS.—It is between this form of glaucoma, optic-nerve atrophy, and cataract that the diagnosis will present most difficulties for the practitioner. The following diagnostic table may be of value:—

GLAUCOMA SIMPLEX.	OPTIC-NERVE ATROPHY.	CATARACT.
Tension increased (at times early in the disease, constantly later).	Tension is normal.	Tension is normal, except in traumatic or complicated cataract.
Episcleral veins are dilated and tortuous.	Normal.	Normal.
Cornea may be hazy and anesthetic.	Normal.	Normal.
Anterior chamber may be shallow.	Normal.	Normal or slightly shallowed.
Iris is normal at first, atrophic later.	Normal.	Normal.
Pupil is slightly dilated early; dilated and fixed later.	Normal or dilated; Argyll Robertson pupil is often present.	Normal.
Pupil gives greenish or "glaucous" appearance.	Pupil is black.	Pupil is whitish or grayish.
Slight pain is sometimes present in early stage.	No pain.	No pain.
Knee-jerks are unaffected.	Often lowered or lost.	Unaffected.
Arterial pulsation is present or easily produced.	Arterial pulsation is rarely present.	No pulsation.
Whole disc is excavated.	Whole disc is concave—saucer-shape.	Disc is normal.
All blood-vessels bend sharply over the edge of the disc. They may be seen indistinctly at the bottom of the excavation, and can be brought to view by using a weaker convex or stronger concave glass in the ophthalmoscope.	Blood-vessels pass from disc into the retina without making sharp bend or curve. They are smaller than normal.	If the opacity in the lens does not prohibit the use of the ophthalmoscope, the vessels appear normal. They may look blurred in one meridian and clear in another, from astigmatism.
Color-fields are commensurate to form-fields.	Disproportionate loss.	

Oftentimes years elapse before the disease ends in absolute glaucoma, or a case of glaucoma simplex may suddenly show acute inflammatory symptoms and soon the little vision remaining is destroyed. Late in the history of glaucoma simplex anyone can make a diagnosis. The eye is hard, the anterior chamber is shallow or obliterated, the pupil is dilated and fixed, the cornea is anesthetic, the lens is cataractous, and the globe is marked by enlarged and tortuous vessels. Pain is now a prominent symptom.

TREATMENT.—Any disease of the general system, and particularly any disease of the nose, from which the patient suffers, must receive proper treatment. Fatigue and worry should be avoided. The patient should obtain sufficient rest, and excesses of all kinds should be avoided. As re-

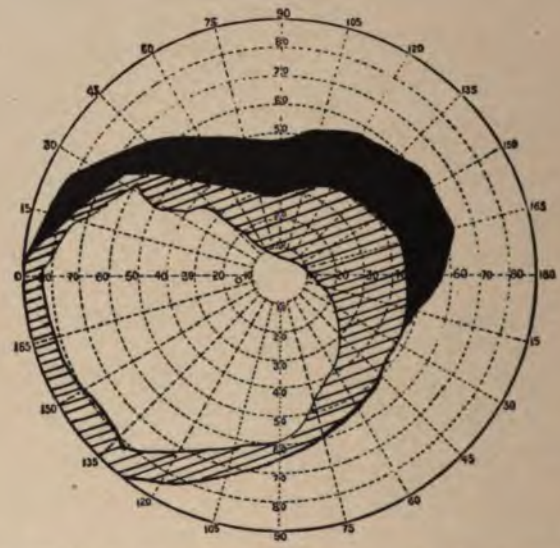


Fig. 345.—Field of vision of left eye in a case of glaucoma simplex. (AUTHOR.)

The black area shows the contraction discovered at the first examination; the shaded area shows the further loss found six months later.

gards the local remedies,—such as pilocarpin, eserin, etc.,—which some ophthalmologists use month after month and year after year in cases of glaucoma simplex, the author wishes to enter a protest against their use except as mere temporizing agents. Many cases of blindness result from this form of glaucoma, the patient and physician wasting valuable time in using miotics. The period during which operative treatment might have done good is allowed to pass. The author is firmly of the opinion that every case of glaucoma simplex possessing vision equal to the counting of fingers at one foot or more should be given the benefit of an operation. The earlier an iridectomy is performed, the greater will be the probability of a favorable result. It is only fair to say, however, that, in cases of glaucoma

simplex with marked contraction of the visual field, great reduction in visual acuity, palpable increase of tension, and deep excavation of the nerve-head, many eminent ophthalmologists regard iridectomy as contra-indicated. In such advanced cases it often occurs that iridectomy does not check the glaucomatous process and the patient soon becomes blind. In cases in which vision is on some days normal and others foggy, with beginning limitation of the field, an iridectomy should be made as soon as the diagnosis is certain. If the result of this is to relieve the symptoms, and vision does not become less, the patient is dismissed with the injunction to return at once on the

Fig. 346.—Section of superior cervical ganglion in chronic irritative glaucoma. (AUTHOR.)

(Original drawing by DR. CARL FISCH.)

1, Artery. *2*, Vein. *3*, Area of small round-cell infiltration. *4*, Normal nerve-cell. *5*, Connective tissue. *6*, Atrophied nerve-cell. (Leitz: ocular 4, objective 6.)

advent of the old symptoms. If the iridectomy does not check the progress of the disease, the author resorts to sclerotomies and later, if necessary, removes the superior cervical ganglion of the sympathetic nerve of the same side. Miotics can be used at any time, but it is wrong to let glaucoma progress without exhausting all the therapeutic resources at our command. The question may be asked: "If excision of the sympathetic is so valuable in glaucoma simplex, why make an iridectomy?" The answer is this: The benefits to be obtained from an excision of the superior cervical ganglion are greater in an eye from which part of the iris has been excised.

Miotics are not to be entirely discarded, but their use should be limited chiefly to tiding the patient over the short period during which he can make arrangements for operative treatment. If the patient refuses operative treatment, a miotic should be prescribed. The best of these agents is arecolin. It is used in a $^1/_2$- to 1-per-cent. strength solution. Eserin and pilocarpin are also valuable miotics. Gentle massage, by the hand or by the ophthalmo-oscillator, employed twice a day, is sometimes followed by improvement of vision and deepening of the anterior chamber.

It must not be expected that any treatment will cure all cases of glaucoma. However, the plan of treatment here outlined will show much better results than will the interminable use of miotics. These agents are useless unless they contract the iris and lower the tension. They are without practical value in an old case of glaucoma with an atrophic iris. Eserin, when used alone, often causes congestion of the ciliary processes, possibly leading to increase of tension. If used in conjunction with cocain, the result is miosis, and, in favorable cases, reduction of tension occurs without congestion of the ciliary processes. When employed, eserin "should be used in the minimum amount and with the minimum frequency which suffice to contract the iris and keep it contracted" (Bull). The salicylate and hydrobromate of eserin are preferred to the sulphate or hydrochlorate (Bull). Pilocarpin is said to lower the tension in some cases of glaucoma in which eserin fails. As Lilienfeld has shown, it may contain an impurity known as jaborin, an isomer of pilocarpin, which acts as a mydriatic. Hence, pilocarpin preparations should be subjected to physiologic tests before being sold.

Chronic Irritative Glaucoma (Chronic Inflammatory Glaucoma).—This form of glaucoma is distinguished from the acute variety by the constant presence of characteristic symptoms, which, during periodic exacerbations, become more marked. It presents prodromal symptoms. After a time the glaucomatous crises appear at shorter and shorter intervals. The eye remains hard. The cornea is steamy and opaque. The anterior ciliary veins appear dilated upon the bluish-gray sclera. The iris ceases to react to light; the pupil becomes dilated *ad maximum*, and finally the iris is reduced to a narrow band covered in large part by the conjunctival limbus. In advanced cases the iris loses its normal markings, its stroma disappears, and the pigment epithelium becomes visible. The cornea is anesthetic. The anterior chamber is shallowed by the forward movement of the lens. Pain of greater or less severity is an almost constant symptom. At intervals the steaminess of the cornea disappears, permitting an ophthalmoscopic examination. This shows the retinal veins to be dilated and tortuous, the arteries narrowed and pulsating either spontaneously or on slight pressure, and the optic papilla excavated. There is progressive reduction in visual acuity and progressive contraction of the visual field. The disease finally ends in complete blindness. Pain, however, persists, and makes the patient clamor for relief.

TREATMENT.—This form of glaucoma calls for an iridectomy. If this fails to reduce the tension, the superior cervical ganglion of the sympathetic may be excised or cyclicotomy may be tried.

Acute Inflammatory Glaucoma.—Although this disease may appear suddenly, it is the rule that certain premonitory signs are known to have been present. They are often overlooked or are disregarded by the patient, to such an extent that the examiner must ask many questions to obtain the true history of the case. A patient who is about to be subject to an outbreak of glaucoma will probably have had days of misty or foggy vision. He may have seen a rainbow-like appearance around a light; and these symptoms may have been more noticeable after loss of sleep or in the presence of great fatigue or strong emotional excitement. He may have changed his glasses more often than is usual for presbyopic persons. These symptoms appear from time to time. The intervals between them may shorten and the condition known as glaucoma simplex may develop; or inflammatory symptoms may arise, and the case is then one of acute glaucoma. The exciting cause of an outbreak may be great weakness induced by fatigue, hemorrhage, or shocking news. Whatever the cause, the patient experiences excruciating pain in and around the eye. The sight is diminished and may be entirely lost in twenty-four hours, in the fulminant form of the disease. The conjunctiva and lids are swollen, and the eye is intensely red around the corneoscleral junction. The anterior chamber is shallow, the cornea is anesthetic, and the eyeball shows tension of + 2 or 3. The patient has fever and is restless. Often vomiting is a prominent sign, and it may lead to the belief that the case is one of sick headache or stomach trouble. Pain, being so prominent a symptom, may cause the practitioner to think he is dealing with iritis—an unfortunate diagnosis, resulting in loss of the eye.

DIAGNOSIS.—The diagnosis of acute inflammatory glaucoma can be made with the fingers. The increase of tension should be evident to any physician capable of the intelligent use of the sense of touch. The examination is to be conducted as described in the chapter on examination of the eye. If the media are clear, the ophthalmoscope may or may not show the characteristic excavation of the head of the optic nerve. In some cases the vision is lost so rapidly that blindness ensues before excavation appears. In some cases of acute inflammatory glaucoma the head of the optic nerve appears swollen.

TREATMENT.—This form of glaucoma usually yields to a properly executed iridectomy. If iridectomy fails to relieve the tension, the surgeon may employ a posterior sclerotomy or a cyclicotomy. If the case still progresses unfavorably, sympathicectomy may be done.

Hemorrhagic Glaucoma.—Intra-ocular hemorrhages occur in glaucoma. They may appear late in the history of the case—glaucoma with hemorrhages; or, as is sometimes observed, hemorrhages appear in an apparently normal eye, and later the tension rises (hemorrhagic glaucoma). It is

necessary to draw clearly a distinction between these conditions, not only for prognostic, but also for legal, reasons. An operation which might be undertaken in an old case of glaucoma with hemorrhages would be entirely valueless in hemorrhagic glaucoma.

Hemorrhagic glaucoma may be defined as an ocular disease, with increased tension, following a previous hemorrhagic retinitis. As Stirling says, it "must be distinguished from hemorrhage into an already glaucomatous eye"—a valuable point to which Risley first directed attention. It is fortunately a disease rarely met with, Risley and Oliver having found it but 12 times among 60,000 ophthalmic cases.

Symptoms.—The patient usually seeks relief on account of failing vision due to intra-ocular hemorrhage, which is a result, as a rule, of some gross change in the vascular system (endarteritis or thrombosis). The symptoms of a subacute or of an acute glaucoma follow the hemorrhages at an interval of weeks or months. The ophthalmoscopic signs, where the media are clear, consist of tortuosity of the retinal vessels, and the presence of numerous flame-shaped hemorrhages, which are particularly abundant in the macular region. The optic disc may be red and hazy, or it may show pronounced neuroretinitis. The retinal arteries are usually small and the veins are large. Later in its history the eye shows the nerve-head deeply cupped and filled with lymph and blood.

Etiology.—As regards age, most cases occur between 50 and 70 years. Weinbaum saw a case 26 years old and the author met with one, a young man, aged 25 years. The disease usually affects one eye only, although cases have occurred in which both were involved. The most constant of the general changes found in cases of hemorrhagic glaucoma is a general arteriosclerosis, which may be a part of or separate from general senile changes. The disease may follow the ocular changes of albuminuria. Treacher Collins considers that there is an analogy between hemorrhagic and primary glaucoma—a view which is opposed by many ophthalmologists. Alt considers that hemorrhagic glaucoma is always caused by hemorrhages, and that hypermetropia is behind the trouble. Noyes saw cases due to, or at least associated with, embolism of the central retinal artery. Richey believes that the disease depends on disease of the vessel-walls which are too weak to bear increased arterial tension. Risley states that "it is but a local expression of the disease of the general vascular tree, and as such anticipates in blindness the fatal result which in many cases speedily follows."

Pathology.—The pathologic changes of hemorrhagic glaucoma are located in the eyeball and in the cervical portion of the sympathetic nerve. The ocular changes comprise inflammatory and degenerative lesions in the chorioid, iris, and ciliary body. In some cases the chief lesions are in the retinal vessels. The changes consist of "sclerosis, hyalin changes in the vessel-walls, obstruction of the lumen by thrombi, distension of the vascular canals with blood, and ruptures. With these, the signs of more or less

gross inflammatory reactions in the surrounding tissues are constantly found" (Oliver). The cornea may show bullæ. The lens often is cataractous and the vitreous humor is shrunken. There are hemorrhagic extravasations into the retina, which is usually detached and cystic. In cases where the process has long continued the cornea becomes ulcerated and the place of the vitreous is taken by grumous and gelatinoid exudates. Such eyes end in atrophia bulbi or in panophthalmitis.

Fig. 347.—Section of superior cervical ganglion of the sympathetic nerve in a case of hemorrhagic glaucoma. (AUTHOR.)

(Original drawing by DR. CARL FISCH.)

1, Pigmented nerve-cell. *2*, Transverse section of non-medullated nerve-fibres. *3*, Network of intracapsular nerve-fibres. *4*, Nerve-cell process. *5*, Unpigmented nerve-cell. *6*, Non-medullated fibres. *7*, Nuclei of perineurium. *8*, Vein. *9*, Polynucleated leucocyte. *10*, Zone of round-cell infiltration. *11*, Mast-cell. *12*, Artery. *13*, Medullated fibres from motor spinal nerve. (Leitz: ocular 4, objective 6.)

In a case of hemorrhagic glaucoma observed by the author the pathologic changes of the eye could not be studied except ophthalmoscopically because an enucleation was not made, the excision of the superior cervical ganglion of the sympathetic nerve having rendered an enucleation unnecessary. Sections of the ganglion show marked pathologic changes, as can be seen by comparison of Figs. 339 and 347.

PROGNOSIS.—The prognosis of hemorrhagic glaucoma is unusually grave. The disease heretofore has been considered not only fatal to sight, but in the large majority of cases removal of the globe has been necessary.

In 28 eyes whose history was collected by Schweigger, vision was lost in all, and 24 were finally enucleated. Iridectomy, opticociliary neurectomy, and sclerotomy have been followed in almost all cases by hemorrhage requiring immediate removal of the globe or by a return of violent pain necessitating the same procedure. Recently, however, Oliver has recorded a more favorable prognosis from iridectomy performed slowly under general anesthesia, the patient being placed on a bed with the head much higher than the feet, so as to diminish the chances of increased blood-pressure. This surgeon, as a result of careful attention to his cases of hemorrhagic glaucoma, was able to save for three of eight cases almost normal vision for eight, six, and four years, respectively. The fields of vision in these cases are fairly large. In two other eyes of this series the disease was held in check for several months, the patients having useful vision, but ended in further hemorrhages due to trauma. Two cases of hemorrhage into the fellow-eye, previously operated on, were followed by a permanently successful iridectomy on the second eye, the first eye being enucleated.

Although Oliver's cases show an unusually successful result from slowly performed iridectomy in hemorrhagic glaucoma, the author believes with Abadie that the operation of sympatheticectomy probably offers a better chance of improvement than any operation which can be made on the eyeball.

TREATMENT.—General treatment is required in every case. The patient's surroundings should be cheerful and hygienic. He should obtain sufficient sleep. All causes of excitement are to be avoided, and alcoholic beverages are to be prohibited. The internal administration of salicylate of sodium is useful. Abadie advises the administration of quinin and ergot. Search should be made for gouty, rheumatic, or syphilitic manifestations, and, if found, proper treatment should be instituted.

As regards operative treatment, the choice lies between iridectomy and sympatheticectomy. Such questionable procedures as paracentesis of the cornea, anterior or posterior sclerotomy, scleral trephining, and stretching the external nasal nerve are out of place. Electricity is not indicated in this disease.

Glaucoma Absolutum.—This term is applied when the glaucomatous eye is completely blind. The eye is then of stony hardness. The pupil, widely dilated, is of a greenish or dirty-gray color. The anterior chamber is almost completely abolished. The cornea is clear, but insensitive; and the distended anterior ciliary veins form a prominent network about the globe.

At a later period, such an eye undergoes *glaucomatous degeneration.* The cornea becomes opaque, the lens cataractous, the sclera ectatic, and after a long period the globe undergoes softening and atrophy. It may develop an ulcus serpens of the cornea, with sequent iridocyclitis or panophthalmitis. Ribbon-like opacity of the cornea is frequent in eyes undergoing glaucomatous degeneration. During much of the period of glaucomatous

degeneration the patient has subjective luminous impressions, which delude him into the belief that his vision will some day be restored.

TREATMENT.—If the glaucomatous eye is entirely blind, it will demand no treatment so long as it is quiet. Unfortunately, however, such eyes usually become very painful and the patient clamors for relief. Medicines are valueless in this condition. Heretofore the operations for the relief of pain in absolute glaucoma have been iridectomy, neurectomy, and enucleation. Of these, iridectomy may be tried. Often, however, after making an iridectomy or an excision of the optic and ciliary nerves, the pain will return. The disadvantage of enucleation is that it causes a great deformity. A posterior sclerotomy may relieve the tension and pain. Excision of the superior cervical ganglion of the sympathetic nerve will relieve the pain of absolute glaucoma for a long period, and often enables the patient to retain the eyeball. In the stage of glaucomatous degeneration only enucleation is valuable.

Fig. 348.—Acquired hydrophthalmos. (AUTHOR.)

The specimen, which is from a girl aged 15 years, shows complete adhesion of the pupillary margin of the iris to the lens, which is shrunken and displaced. The cornea is much enlarged, and through the greater part of its extent the iris is adherent to it. The iris is also atrophic. This eye measures: vertical diameter, 22 millimetres; axial diameter, 30 millimetres.

Treatment of the Apparently Unaffected Eye in Glaucoma is a matter of importance. Careful examination will frequently show that the apparently normal eye presents premonitory signs of glaucoma, in the way of transient increase of tension, obscuration of sight, slight steaminess of the cornea, etc. In such cases, although vision may be for most of the time normal, the author advises an iridectomy, which is to be made as soon as the eye first operated has recovered. This advice is especially to be urged upon patients who live far from competent ophthalmic surgeons.

Glaucoma after Cataract Operation is discussed in the chapter on diseases of the crystalline lens (page 458).

Prevention of Primary Glaucoma.—If errors of refraction and accommodation were properly treated early in life, it is highly probable that the percentage of glaucoma cases would be much reduced. If a patient already has glaucoma, he should be urged to lead a regular and quiet life. Causes of

excitement should be avoided. Stimulants should not be used. Previous to making an iridectomy or any other operation upon one eye a miotic should be instilled into the other eye, to prevent an attack of glaucoma.

Hydrophthalmos (Buphthalmos; Infantile Glaucoma; Congenital Glaucoma).—This is a rare disease of childhood, which is characterized by increased intra-ocular tension and enlargement of the eyeball in all of its diameters. The increase of tension is due to blocking of the drainage apparatus. Some writers speak of this disease as always congenital, but this is a mistake. The author has seen two cases in which the disease was acquired. In one it followed gonorrheal ophthalmia, in the other an injury. The latter case was that of a girl of 15 years, who, at the age of five years, was struck with the tine of a table-fork, which pierced the cornea and was followed by iris-prolapse. A surgeon desired to enucleate the eye, but the parents would not consent. The patient was lost sight of and ten years later came with pronounced hydrophthalmos. A section of the enucleated eye is shown in Fig. 348. How came it that this case was transformed from one of iris-prolapse into hydrophthalmos? Shortly after the receipt of the injury the wound closed and the aqueous humor separated the iris from the corneal wound. An iritis followed and, being untreated, adhesions fastened the iris to the lens-capsule, thus completely separating the posterior from the anterior chamber; and gradual accumulation of aqueous humor raised the intra-ocular pressure, with the result that the eye became enlarged. This could occur only in a child, since in the adult the sclera is unyielding except it be softened by inflammation.

For anatomic reasons, the disease tends toward the production of grave changes in all the structures of the eye. In response to increased intra-ocular tension, the eyeball increases in every diameter. The cornea becomes enlarged to such an extent that the term keratoglobus can be applied with propriety. The lens, which is often rudimentary, is arrested in its growth; and the eyeball from being hypermetropic, or possibly emmetropic, in the acquired form of buphthalmos becomes greatly myopic. Vision is gradually reduced by excavation of the optic-nerve head or by chorioidal and retinal changes. The globe protrudes to such an extent as to cause a hideous deformity. The eye shows signs of irritation. It is subject to frequent attacks of conjunctivitis and keratitis. The cornea becomes thin, and often looks bluish from contact of the adherent iris. The line of demarcation between the cornea and sclera is lost. The ciliary region often presents nodules. The anterior chamber may be of great depth in the centre and be obliterated at the periphery. Often the iris is atrophic and presents numerous rents. Generally a view of the depths of the eye cannot be had, because of opacities in the lens or the presence of exudation in the pupillary area.

Cases of buphthalmos sometimes go on to spontaneous cure. Thus the increased tension may expand the eye up to a certain point, at which the enlargement becomes stationary, and the tension may become normal.

Although such an eye remains large and the sight is defective, no further increase occurs and vision is not further impaired.

Etiology.—The causes of acquired hydrophthalmos are trauma and perforating corneal ulcers. As regards the cause of the congenital form, there is much doubt. Johnson reports the occurrence of the disease in three children in the same family. A failure of the iris to separate from the posterior surface of the cornea at its extreme periphery can occur unassociated with anterior synechia elsewhere, and this offers an easy explanation of congenital buphthalmos; but why this separation should not take place is not known. Collins has made microscopic examinations of several eyes afflicted with congenital glaucoma, and has found that in all the angle of the anterior chamber was closed by adhesion of the root of the iris to the cornea. The canal of Schlemm may be absent.

Fig. 349.—Binocular hydrophthalmos following ophthalmia neonatorum. (Author.)

Diagnosis.—Hydrophthalmos may be mistaken for interstitial keratitis. These diseases present somewhat similar appearances in their initial stage. The differentiation can be made by the state of the tension, which is increased in hydrophthalmos.

Prognosis and Treatment.—Iridectomy in this disease is rarely followed by improvement. During the operation the zonula is likely to become ruptured and there is loss of vitreous humor. Bergmeister has recorded the case of a child, aged 13 years, on whom he made an iridectomy for congenital hydrophthalmos when the patient was six months old. A cure followed the operation. Stölting has had favorable results from repeated sclerotomies. Snellen treats these cases by paracentesis of the anterior chamber. Sympatheticectomy has been tried in a few cases without improvement. The use of miotics is of little, if any, value. When the eye is greatly enlarged, thus causing a deformity, and vision is lost, enuclea-

tion or a Mules operation is to be advised. Such eyes are sources of both physical and mental suffering.

SECONDARY GLAUCOMA.

This term is applied to all cases in which increased intra-ocular tension comes from other pathologic processes. It may be acute or chronic. Among the chief causes of secondary glaucoma are: (1) a neglected iritis, leading to closure of the aqueduct from the posterior to the anterior chamber: (2) incarceration of the iris in a corneal or scleral scar; (3) ectasiæ of the cornea and sclera; (4) iridocyclitis, which, as a rule, leads to only temporary increase of tension; (5) closure of a corneal fistula; (6) rapid swelling of an injured lens; (7) dislocation of the lens; (8) hemorrhages into the retina; (9) the growth of intra-ocular tumors; (10) chorioiditis and high myopia. These conditions are all considered elsewhere in this treatise, and do not call for further consideration in this place.

HYPOTONY.

Hypotony is a diminution of intra-ocular tension, and indicates a reduction in volume of the contents of the eyeball. It follows perforating wounds or ulcers, and occurs in cases of atrophy of the globe after iridocyclitis. It is found after injury to the cervical portion of the great sympathetic nerve or after removal of its ganglia. Slight reduction of tension follows the use of a bandage which has been tightly applied to the eye, and is a common symptom in cases of keratitis. The local use of cocain causes slight hypotony. Dionin is said to produce a similar effect.

OPERATIONS FOR GLAUCOMA.

Of the many operations which have been proposed for the cure of glaucoma only three or four are worthy of mention. They are iridectomy, sclerotomy, cyclicotomy, and excision of the superior cervical ganglion of the sympathetic nerve. Not one of these procedures can be depended upon to cure all cases of glaucoma. It will be found advisable in some cases to employ two of these operations at different periods, or it may be necessary to make an iridectomy twice on the same eye, to control the tension. Of the operations mentioned, iridectomy, introduced by von Graefe in 1857, has long held sway as the chief treatment for glaucoma. Sclerotomy and cyclicotomy have never attained the popularity which has been accorded to von Graefe's operation. Recently, in 1897, Jonnesco performed an operation for glaucoma which had been already suggested by Abadie, viz.: excision of the superior cervical ganglion of the sympathetic nerve. The favorable results obtained by Continental surgeons—Jonnesco, Abadie, Réclus, Gérard-Marchant, Chauffard and Quénu, Jeunet and Bled—in the treatment of glaucoma by this procedure led the author to adopt the opera-

tion early in its history. Unfortunately in many of the cases treated by this method the ultimate results have not been satisfactory.

Iridectomy.—An iridectomy for glaucoma is a much different operation from an excision of the iris for an optical purpose. The latter requires the removal of a small part of the iris through an incision made in the corneal tissues. The former contemplates the excision of two-fifths of the iris-tissue through an incision made in the sclera close to or through the canal of Schlemm. The amount of iris-tissue removed is of less importance than is the site of operation. The object is to restore the drainage of the anterior chamber. The instruments required are a speculum, fixation-forceps, bent keratome or the narrow von Graefe cataract-knife, iris-forceps, scissors, and spatula. While the iridectomy for an optical purpose can often be made under cocain or holocain anesthesia, the operation for glaucoma usually requires general anesthesia for its successful execution. Whenever possible the excision is to be made upward so that the upper lid may cover the coloboma. A more important rule is to excise that part of the iris which is least adherent to the cornea: *i.e.,* the incision is made opposite the deepest part of the anterior chamber. The ordinary precautions for surgical cleanliness having been observed, the operator introduces the speculum, fixes the globe, and passes the keratome at a point one millimetre behind the limbus. The instrument is steadily pushed into the anterior chamber, making a wound five or six millimetres in extent. As the keratome is passing into the eye the handle must be depressed sufficiently to prevent injury to the iris and lens. This often is a difficult procedure, owing to the shallowness of the anterior chamber. In such cases—*i.e.,* where the anterior chamber is very shallow—a scleral puncture should first be made with a von Graefe knife at a point five to seven millimetres behind the cornea. This procedure is followed by a sinking of the lens and a consequent deepening of the anterior chamber. The keratome must be withdrawn slowly in order that the reduction of intra-ocular pressure may not occur too rapidly and cause hemorrhage from the retinal vessels. The iris is drawn out of the eye and cut at one extremity of the scleral incision. Another cut is then made at the opposite end of the wound, and, last, the piece of iris to be removed is to be cut off as close to the base as possible. The angles of the iris-coloboma are to be freed by the spatula. It is of the greatest importance that the iris shall not become entangled in the wound. An ordinary gauze dressing and bandage are applied to both eyes. The dressing is changed the next day, at which time 1 drop of a 1-per-cent. strength solution of atropin is used for the purpose of preventing adhesion of the iris to the lens-capsule (Noyes). *This is the only place and time in a case of glaucoma in which atropin should be used.* The after-treatment of the case is usually uneventful.

Iridectomy in the Aphakic Eye.—Here a blunt hook is used to draw the iris out into the wound, forceps frequently failing to grasp it. Commonly a bead of vitreous will be lost, since in most cases of glaucoma which

appear in the aphakic eye there is vitreous humor in the anterior chamber. The iris, drawn out by the hook, is held with forceps and excised. The spatula is used as mentioned before. Recovery is generally uninterrupted.

ACCIDENTS.—Any one of several accidents may occur during an iridectomy for glaucoma: (1) the surgeon may pass the keratome too far forward, thus failing to open the canal of Schlemm, or (2) too far backward, injuring the ciliary body or lens; (3) failure to tilt the keratome forward after penetrating the cornea may cause the point of the instrument to injure the iris or lens-capsule; (4) sudden withdrawal of the instrument, by rapidly lowering intra-ocular pressure, may cause rupture of a retinal vessel; (5) prolapse of vitreous may occur; (6) the angles of the iris-coloboma may be entangled in the wound; or (7) ill-directed traction, the use of an imperfect instrument, or too great force used in withdrawing the iris may cause iridodialysis and free hemorrhage into the anterior chamber, thus obscuring a view of the angles of the iris-coloboma. A miotic should not be used immediately preceding an iridectomy, since under such circumstances considerable force is required to draw the iris out of the wound, and iridodialysis may be caused.

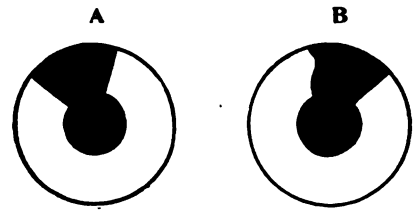


Fig. 350.—Appearance of pupil after iridectomy for glaucoma.

A, The lips of the iris are straight. *B*, The one lip of the iris is incarcerated in the wound.

Sympatheticectomy.—This is a major operation, demanding general anesthesia, scrupulous cleanliness, and an accurate knowledge of anatomy.

The skin from the clavicle to the ear, and from the median line in front to the spine posteriorly, should be shaved, scrubbed, and a bichlorid pack should be applied several hours before the operation. The hair behind the ear should be removed from a surface half the size of the palm. Immediately before the operation the skin should be washed in a strong solution of potassium permanganate, which is followed by a solution of oxalic acid. A bichlorid solution is used as a wash immediately before the operation.

The steps in the procedure are: (1) the incision and separation of tissues down to the vertebral column, (2) identification of the sympathetic nerve, (3) excision of the superior ganglion, and (4) closure of the wound and after-treatment.

1. THE INCISION should be four or five inches in length and should be made always behind and parallel with the posterior border of the sterno-cleido-mastoid muscle. It should begin at a point over the occipital bone

corresponding to the origin of the muscle. The incision generally involves the external jugular vein, which is tied in two places before being cut.

After incising the skin and superficial fascia the deep fascia is reached and the spinal accessory nerve is cut, permitting the operator to separate the sterno-cleido-mastoid muscle completely from the adjacent tissues. The deep dissection is done with the fingers or with a blunt instrument, never with a sharp one. The upper part of the wound can be enlarged by cutting with scissors until the skull is reached. It is important to do this if the surgeon expects to remove the greater part of the superior ganglion. The separation of tissues down to the vertebral column can be done rapidly with

Fig. 351.—Site of the incision for removal of the superior cervical ganglion of the sympathetic nerve. (AUTHOR.)

the fingers, provided the operator follows the intermuscular fascia. The carotid sheath is pulled forward with the sterno-mastoid muscle by a retractor and the trapezius muscle is held backward in the same way, thus permitting an inspection of the deep wound. The landmarks to be followed are the transverse processes of the vertebræ.

2. THE IDENTIFICATION OF THE SYMPATHETIC NERVE is sometimes easy, at other times exceedingly difficult. The variations in the size and situation of the cervical portion of the sympathetic nerve cannot be appreciated by reading text-books on anatomy, but must be observed on the living subject. It is generally stated that the sympathetic nerve is inclosed in a sheath separate from and behind the carotid sheath. This often is not true.

Several times the author has seen the sympathetic lying alongside the pneumogastric nerve, inclosed in the same fascia with the internal jugular vein and the carotid artery. On this account, and for the additional reason that the pneumogastric and sympathetic nerves often are of the same size, it is wise to open the carotid sheath extensively and follow the nerves downward to the point where the middle cervical ganglion is located. This,

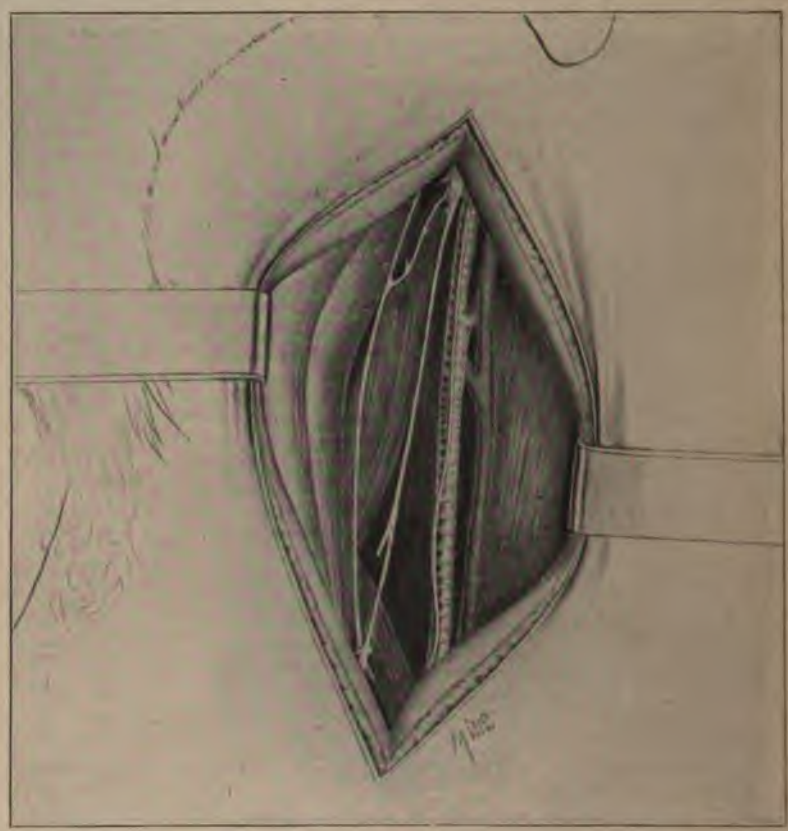


Fig. 352.—Operation for excision of the superior cervical ganglion of the sympathetic nerve. (AUTHOR.)

(Original drawing by DR. R. W. MILLS.)

At the upper part of the wound is the superior cervical ganglion; at the lower extremity is the middle ganglion. Deep in the wound are the internal jugular vein, carotid artery, and pneumogastric nerve.

of course, will settle the question of identification, since the pneumogastric nerve has not a middle ganglion. If the middle ganglion of the sympathetic nerve should be absent, the operator should follow each nerve upward to the farthest possible extent. The spindle-shaped superior ganglionic expansion of the sympathetic serves to identify the nerve to be excised. If this method of identification should fail, the surgeon can pick up each nerve

separately and irritate it, and watch the effect on the heart. The one whose irritation affects the heart should not be excised.

The surgeon cannot depend upon the size of these nerves, since the two have been seen lying together and of the same size and color. The pneumogastric may be so much atrophied that it is scarcely larger than a pin's head, and the sympathetic may be large or small.

3. Excision of the Ganglion.—The nerve having been identified, the next step is to excise the ganglion. The operator first separates the ganglion to the base of the skull; the numerous branches given off from the ganglion are cut; the ganglion is held taut with forceps by an assistant, while the operator places the left index finger under it and cuts the ganglion as high as possible, using strong curved strabismus scissors. The excision is completed by severing the nerve-strand an inch below the ganglion.

4. The Closure of the Wound is done with superficial sutures. It is not necessary to use deep sutures. The after-treatment in the author's cases has been uneventful. The author's first patient was put up in a plaster dressing, which was uncomfortable and unnecessary.

When properly done there is only trifling hemorrhage from this operation. The patient leaves the hospital on the sixth or seventh day. An unpleasant sequel is severe pain located in the neck and shoulder.

Effects of Excision of the Superior Cervical Ganglion.—The effects of removal of this ganglion are immediate and remote. The immediate effects are relief of ocular pain, the production of lacrimation and conjunctival injection, together with a discharge from the corresponding nostril, unilateral sweating, and contraction of the pupil. Often there is an immediate reduction in intra-ocular tension. The pupillary and conjunctival changes are noted within five minutes after the excision.

The remote effects are ptosis, which appears on the third or fourth day, improvement of vision, and in some instances a tardy contraction of the pupil and a tardy reduction of intra-ocular tension. To these there must also be added a slight sinking of the eyeball into the orbit (enophthalmos), and a feeling of heaviness in the head.

Immediate reduction of intra-ocular tension does not always occur. In the author's second case at the end of eight days the tension was + 2. On the sixteenth day it was normal. In the first case reduction of the tension was immediate. The relief from pain in the first case was immediate and lasting. This patient had not been free from pain for two months previously. The slight ptosis following sympathetic ectomy is to be attributed to paralysis of Müller's muscle. Sinking of the eyeball is no doubt due to paralysis of the unstriped peribulbar muscular fibres which are found in Tenon's capsule.

Contraction of the pupil is usually an immediate result; it may, however, appear tardily. Thus, in the author's first case the pupil was unchanged until the fourth day after the operation; and it did not become at any time as markedly contracted as in the other patients. In the

third case—that of optic-nerve atrophy—the pupil was markedly contracted within five minutes after the excision.

Lacrimation, conjunctival injection, and nasal moisture are transient symptoms which are generally absent after the first or second day.

The immediate improvement in vision following excision of the superior cervical ganglion is remarkable; but unfortunately in many cases it is not lasting. It must be stated, however, that the cases in which sympatheticectomy has been performed have been usually desperate ones—cases in which no good result would be expected from iridectomy. It is highly probable that this operation will give more satisfactory results if performed earlier in the history of the case.

Conclusions as to the Value of Sympatheticectomy.—1. Excision of the superior cervical ganglion is a valuable procedure in glaucoma, but is not to be ranked with iridectomy in remedial value. While, in general terms, excision is not to be advised in non-iridectomized eyes, such excision is admissible under the following circumstances: (1) where iridectomy or sclerotomy is refused, (2) in hemorrhagic glaucoma, and (3) in glaucoma simplex with great loss of vision.

2. Sympatheticectomy is of more value in glaucoma simplex than in inflammatory glaucoma.

3. In inflammatory glaucoma, on which iridectomy has been done without benefit, excision of the superior cervical ganglion may be tried.

4. In cases of absolute glaucoma with pain sympatheticectomy may be tried before resorting to enucleation.

5. In unilateral glaucoma excision of the sympathetic ganglion is to be done only on the corresponding side.

6. In the hands of a careful operator excision of the superior and middle ganglia is a safe operation. Of nearly 100 cases of glaucoma in which this operation has been performed, 1 death has been reported, which was due to accidental infection.

7. The post-mastoid route is to be preferred in excision of any part or all of the cervical sympathetic.

8. Whether the curative effects of sympatheticectomy are as lasting as those of iridectomy and cyclicotomy (Hancock's operation) is questionable.

9. It is not yet known whether or not sympatheticectomy can prevent glaucoma.

Large Paracentesis of the Sclerotic with Cyclicotomy (Sclerocyclocotomy; Hancock's Operation).—This operation was performed by Hancock on the supposition that glaucoma is caused by spasm of the ciliary muscle causing a stasis in the intra-ocular circulation. The procedure is as follows: The eye having been anesthetized by cocain, the lids are held apart by the thumb and fingers, which are also used to steady the eye. The surgeon passes a Beer cataract-knife, with its cutting edge downward, into the sclera at the lower margin of the cornea, the incision being made between the

external and inferior rectus muscles. The instrument is quickly passed downward and backward into the vitreous until one-third to one-half of the blade is hidden. In withdrawing the instrument it is slightly turned so that the wound gapes, permitting the aqueous humor and a small amount of the vitreous to escape. Pollak, who practiced this procedure for forty years, says: "A few seconds only are required for this operation. No dressing or any after-treatment is needed. The relief from increased tension and pain is instantaneous. The lens recedes to its normal position, the pressure upon the ciliary processes is removed, the iris being freed from pressure soon resumes its normal place, the spaces of Fontana are gradually opened, and so also is the canal of Schlemm." Those who have performed this operation are so well pleased with the results that it should be more generally employed. It will be advisable, however, to bandage the eye for two or three days after the operation.

The author has tried Hancock's operation twice only, with satisfactory results. The first patient had had a broad iridectomy made several years before for glaucoma. The eye took on an attack of acute inflammatory glaucoma; tension was much increased, and vision was reduced to perception of light. There was intense pain. Within ten minutes after performing Hancock's operation the eye was free from pain and within a week there was restoration of vision to $^{20}/_{30}$. The other patient was an old and feeble woman, with absolute glaucoma, to whom the administration of a general anesthetic would have been dangerous. Hancock's operation was followed by immediate relief of pain and reduction of tension. Six months later the eye was quiet and the tension was normal.

Sclerotomy.—Sclerotomy was introduced by Quaglino for the reason that leading ophthalmologists believed that the beneficial effects of iridectomy in glaucoma were due, not to excision of the iris, but to the cutting of the sclera. Sclerotomy may be anterior or posterior to the attachment of the iris. Some few surgeons prefer anterior sclerotomy to all other operations for glaucoma; others employ it only in case of failure of iridectomy to reduce intra-ocular tension; and others again reserve it for hemorrhagic glaucoma or for the glaucoma following cataract extraction. In general it may be said that sclerotomy has gone out of vogue in the last ten years, and few surgeons now depend on it. Before a sclerotomy is made a miotic is used, and likewise it is employed for several days after the operation.

Anterior Sclerotomy.—The instruments required are a speculum, fixation forceps, a von Graefe knife or a keratome, and a spatula. Iris-forceps and scissors, or de Wecker's *pince-ciseaux,* should be at hand as reserve instruments. There are several methods of performing this operation. Before attempting any one of them a miotic should be used.

1. Quaglino's Method.—A keratome is inserted two millimetres behind the corneoscleral junction, passing as close to the iris as possible, making a wound three to five millimetres in extent. The instrument is withdrawn

slowly so as to avoid prolapse of the iris. Snellen employs this procedure. This operation has not met with the favor which has been accorded to

2. De Wecker's Method.—A narrow von Graefe knife, or a specially made instrument called a sclerotome, is passed transversely across the eye at a point one millimetre behind the corneoscleral junction. The puncture and counterpuncture are enlarged by sawing movements. The knife is slowly removed before the section is complete, thus leaving a bridge of sclerotic between two wounds.

Comparative Value of these Methods.—Sclerotomy with the von Graefe knife is the more difficult of proper execution. It is less easy to control the proper anatomic position of the wound; prolapse of iris is more likely to occur, particularly near the point of counterpuncture; and more definite

Fig. 353.—Posterior sclerotomy. (After CZERMAK.)

scarring of the eye results with the possibility of unpleasant sequelæ. Sclerotomy with a keratome can give a satisfactory result without any trace of the operation remaining.

Accidents.—The iris may be punctured by the knife or keratome. If the lens is uninjured the accident is of no importance. If, after the incision has been made, the pupil is drawn toward the wound, it indicates that the peripheral part of the iris has become incarcerated in the lips of the wound. The iris should be released with a metal spatula. If this cannot be accomplished, it will be advisable to make an iridectomy. If prolapse of iris occurs during the operation an immediate iridectomy will be in order. The healing process not infrequently is not smooth, the formation of an elevated and nodular scar being not uncommon. These scars lead to fistulæ, and may be the cause of prolonged irritation which yields only to an iridectomy.

Some authors regard a bulging scar as a safeguard, the aqueous humor filtering through it and passing under the conjunctiva, thus reducing the intra-ocular tension. Such a condition must be regarded as dangerous.

POSTERIOR SCLEROTOMY, incision of the sclera behind the attachment of the iris, is employed for the following conditions: (1) removal of foreign bodies in the vitreous chamber; (2) the removal of intra-ocular parasites (cysticercus); (3) in detachment of the retina; (4) in glaucoma either as a primary operation or in cases in which iridectomy has failed; (5) as a step preliminary to iridectomy in cases where the anterior chamber is much shallowed; (6) in traumatic hemophthalmos without rupture of the cornea or sclera.

The operation may be simply a puncture for the relief of increased tension, for the cure of detachment of the retina, or to hasten the absorption of blood in traumatic hemophthalmos; or it may be an incision of considerable size, as in the operation of removal of a foreign body or parasite in the vitreous chamber; or it may be an incision for the purpose of dividing the ciliary muscle, as in Hancock's operation for glaucoma. A simple puncture or an incision is made with the von Graefe cataract-knife, while Hancock's operation (sclerocyclocotomy, cyclicotomy) is usually made with the knife of Beer. Accessory instruments are a speculum and fixation-forceps. Precautions having been taken to insure asepsis, under holocain anesthesia the knife is passed through the sclera. Its direction will depend on the purpose of the operation, but usually it will be meridional. Some operators give the wound a curved, an L-, or a T-shape. Generally the incision is placed in the lower external quadrant, between the attachments of the inferior and external recti muscles, and is five or six millimetres long and one centimetre deep. In puncture for detached retina the knife must often be carried to a greater depth. The eye is to be bandaged for a few days and healing is usually without incident.

CHAPTER XVIII.

SYMPATHETIC EYE DISEASES—INDICATIONS FOR ENUCLEATION.

By the term sympathetic eye diseases we understand those pathologic conditions which are produced in the second eye by the internal transmission of an affection previously existing in the first eye. The eye which becomes diseased by reason of a disease of or an injury to its fellow is called the *sympathizer,* while the other is known as the *exciter.* For many years sympathetic eye diseases have been divided into sympathetic irritation and sympathetic inflammation. Regardless of the attempts to efface the difference between these conditions and affirm the existence of a transition state, we must adhere to the essential clinical differences of both affections.

Sympathetic Irritation is an abnormally strong irritability, often united with diminished functional power, which appears in all the centrifugal and centripetal ocular nerves, depending on irritation of the centripetal nerves in the eye primarily diseased. The sympathetic disease ends with the disappearance of this irritation.

Sympathetic Inflammation, or sympathetic ophthalmia, the ophthalmia migratoria of Deutschmann, is a progressive ocular disease attended with hyperemia and exudation, produced by a disease previously existing in the other eye, and not materially influenced in its course by the cure or removal of the exciting eye.

In sympathetic diseases it is probable that the entire nervous apparatus of the exciter is concerned in the transmission of disease to the sympathizer. The condition producing sympathetic diseases is uveitis (iridocyclitis, iridochorioiditis), which is generally caused by one or other of the following conditions: (1) wound of the ciliary region; (2) lodgment of a foreign body in the eye; (3) perforating wound or ulcer with incarceration of the iris in the corneal cicatrix; (4) operations upon the eyeball—a rare cause of sympathetic ophthalmitis; (5) dislocation, wounds, or calcification of the lens; (6) intra-ocular tumors when causing iridocyclitis; (7) intra-ocular cysticercus (the cause in a few cases); (8) ossification of the chorioid and ciliary body; (9) various rare causes, such as tattooing of the cornea, herpes zoster ophthalmicus, symblepharon, and the wearing of an artificial eye; (10) incarceration of the stump of the optic nerve in the cicatrix following enucleation; (11) blows on the eye without external wound (Mackenzie, Knapp, Schirmer, and Bronner); (12) blows, with subconjunctival rupture of the sclera; (13) in one case a burn by lime was followed by sympathetic ophthalmitis (Noyes).

In case the exciting eye has not been removed, its condition is generally that of beginning or complete phthisis bulbi. In the study of sym-

pathetic eye diseases an important question is this: Are sympathetic irritation and sympathetic inflammation different stages or degrees of the same process, or are they two entirely different processes? Although many authors believe that irritation is an early stage of sympathetic inflammation, there are so many well-authenticated cases where sympathetic inflammation suddenly appeared without signs of irritation that we must hold strictly to the non-identity of these affections. Hence the great importance of enucleating the injured eye without waiting for the advent of sympathetic irritation in its fellow.

SYMPATHETIC IRRITATION.

Sympathetic irritation (sympathetic neurosis) is a functional disease presenting a great variety of symptoms referable to the ocular nerves and manifested by increased excitability and diminished functional power. Whether the affection is more frequent in neurasthenics than in persons with normal nervous systems is a mooted point. The frequency of sympathetic irritation cannot be determined accurately, since its manifestations are often overlooked by both surgeon and patient in their anxiety for the injured member. Pain in and around the eye, radiating through the ciliary branches of the trigeminus, is a common symptom, the periorbital and frontal branches being often involved. It is rarely the case that the pain involves the whole trigeminal distribution. In some cases there is a feeling that the head is incased in an unyielding band. In other cases the pain is described as sticking or boring. These ciliary neuralgias must not be confounded with pain radiating from the injured eye. The pain of sympathetic irritation, like the other symptoms, promptly subsides on removal of the exciting eye. Other prominent symptoms are photophobia, blepharospasm, diminished accommodative power, contraction of the visual field, muscæ, photopsia, diminished visual acuity, pericorneal injection, hyperemia of the fundus, and tenderness over the ciliary region. Spasm of the ciliary muscle and spastic miosis are rare symptoms. The eye tires easily and waters when used for near work or on exposure to light. While a slight redness or haze of the optic papilla may be present, careful examination fails to show any signs of intra-ocular inflammation.

Etiology.—Sympathetic irritation can be produced by various causes, such as insignificant injuries; loss of corneal epithelium; the lodgment of foreign bodies in the cornea or under the upper lid; staphyloma of the cornea and sclera; iridocyclitis; luxation of the lens, especially if it is calcified; rapid swelling of the lens after discission or trauma; or the lodgment of foreign bodies within the eyeball. In all these cases a slight degree of sympathetic irritation may be readily overlooked. Patients with sympathetic irritation often present a shrunken globe, which may be due to external causes (traumata), to a perforating ulcer, or to a spontaneous ocular inflammation. The interval between the involvement of the first eye and the advent of sympathetic irritation in its fellow varies from a few seconds

to a period measured by the life of the individual. In the former case a foreign body lodging in the cornea is followed immediately by sympathetic irritation, while in the latter instance a period of quiescence lasting thirty or forty or more years is ended by the occurrence of pain, photophobia, and pericorneal injection in the good eye. In such cases anatomic examination of the enucleated globe will often show ossific deposits in the shape of a shell, corresponding to the chorioid coat, or the presence of minute spiculæ of irregular form. Not rarely do we find sympathetic irritation after enucleation of the injured eye, or following the placing of a glass or metallic globe in Tenon's capsule, or in the sclera according to Mules's operation. Sympathetic irritation may even occur after enucleation, where the stump of the optic nerve is involved in adhesions and the conjunctiva is not markedly retracted. It also occurs as the result of wearing an improperly fitting artificial eye. In all these cases the cause of sympathetic irritation rests in irritability of the ciliary nerves.

Diagnosis.—The diagnosis must concern (1) the existence of a sympathetic disease and (2) the differentiation between sympathetic irritation and sympathetic ophthalmitis. As regards the first contention, while sympathetic disease has no pathognomonic symptoms, disturbance of vision, the presence of pain in and around the eye, and the occurrence of pericorneal injection, photophobia, and blepharospasm or iridocyclitis appearing in the second eye as early as three or four weeks, or within four months after an injury to its fellow, will point to sympathetic disease. Occurring after the fourth month, such symptoms leave the diagnosis somewhat uncertain. However, in case of doubt it will be best to consider the case one of sympathetic disease until disproved. Schirmer states that the diagnosis of sympathetic inflammation cannot be made with absolute certainty, since identical symptoms may be due to other causes. The differentiation between sympathetic irritation and sympathetic ophthalmitis, which is of the greatest importance in prognosis, will perhaps be simplified by attention to the following table:—

Sympathetic Irritation.	Sympathetic Inflammation.
May appear in a few minutes or many years after injury.	Minimum interval known is two weeks; usually appears within three months after injury, but may be delayed for years.
Pain, photophobia, photopsia, blepharospasm, lacrimation, hyperemia of the conjunctiva, obscuration of vision, weakness of accommodation, and abnormal pupillary action are present.	Sympathetic inflammation may be preceded by these irritation symptoms, but often they are absent.
Objective signs are absent except in some cases the optic-nerve head appears hazy.	Objective signs are those of definite inflammation: plastic or serous iritis, keratitis punctata, and pericorneal injection. Papillitis may be absent, or present as a neuroretinitis or with retinal hemorrhages or edema.

While the diagnosis is usually not difficult, it must be remembered that cases are on record in which the conditions overlapped. Impaired vision in the sound eye after an injury to its fellow may indicate simply sympathetic irritation; but the fundus should be examined for slight neuritis, which, if present, will indicate the onset of sympathetic inflammation. Such a case occurring in the author's practice was cured by removal of the exciter and a long course of atropin and mercurials.

Prognosis.—Almost invariably proper treatment is followed by cessation of all irritation symptoms in this disease. In cases where the patient will not submit to operative intervention attacks of sympathetic irritation may recur at intervals for years without further damage; or, on the other hand, the eye may develop sympathetic ophthalmitis and be lost. It is impossible to foretell with certainty what kind of damaged eye will cause sympathetic disease or what will be exempt. Nor can the surgeon say what interval will elapse between the receipt of an injury and the development of sympathetic irritation. It is because of this uncertainty that all cases, in which sympathetic irritation or inflammation may be expected to develop, should be referred by the general practitioner to a competent ophthalmic surgeon without delay.

Treatment.—In sympathetic irritation the first indication is to remove the source of trouble; and this presupposes an exact diagnosis of the condition of both the exciter and the sympathizer. Removal of foreign bodies—whether in the cornea, conjunctiva, or elsewhere—will be in order. A luxated lens comes in this category. Mooren relieved a sympathetic irritation of two years' duration by removal of such a lens. If the cause of irritation is a corneal staphyloma, it must be removed. If the ectasia is partial, or if posterior synechiæ are present, an iridectomy will suffice. If sympathetic irritation develops in a patient whose other eye has been removed, it will be necessary to submit the stump to the most careful scrutiny without local anesthesia. If exuberant granulations, swelling, and secretion are present, the inflamed patch should be excised or else should be touched with the solid stick of nitrate of silver. If the glass eye used by the patient is defective, it must be removed and a suitable one provided in its stead. If the conjunctiva is drawn backward, and is adherent to the stump of the optic nerve, a further resection of the nerve down to the optic foramen will be necessary.

By far the largest and most important group of cases is that in which a blind or phthisical eye has caused sympathetic irritation. Such a globe will be tender on pressure, and should be removed either by enucleation or evisceration or else evisceration combined with the insertion of an artificial vitreous (Mules's operation). The value of these procedures will be discussed later on in this chapter. Such operations as section of the ciliary nerves and opticociliary neurotomy seem to the author to be inadequate in this condition.

SYMPATHETIC OPHTHALMITIS.

This disease begins insidiously, either with or without signs of sympathetic irritation. It pursues a persistent and destructive course. Once inaugurated, it rarely is checked by treatment. It is one of the most formidable and obscure of ocular affections. While no age is exempt, the disease is much more liable to occur in children before or about the period of puberty. Any one of the conditions mentioned above as likely to cause sympathetic irritation may produce sympathetic ophthalmitis. The most frequent cause of sympathetic inflammation is perforating wounds, which do not heal readily, but undergo chronic inflammation. Only uveal inflammation caused by bacterial infection is capable of producing sympathetic inflammation (Schirmer). Splinters which have entered the eye aseptically never cause sympathetic inflammation (Schirmer). There is no relationship between the severity of the pathologic process in the exciter and the disease in the sympathizer. Often a slight cyclitis in one eye will be followed by complete disorganization in the other. Not infrequently does it occur that the exciter possesses useful vision both during and after the attack of sympathetic ophthalmitis. The disease commonly appears within two or three months after the injury to the fellow-eye, but it may commence in two or three weeks. On the other hand, of 211 cases recorded by the committee of the Ophthalmological Society of the United Kingdom, 12 eyes became inflamed as late as twenty years after injury, and in 3 cases the interval was thirty-seven, thirty-eight, and thirty-nine years, respectively. It has been asserted that sympathetic ophthalmitis has been known to occur as early as the seventh day, and in Alt's table of 110 eyes in 3 it appeared within eight days, but Schirmer states that the minimum interval in cases carefully observed is fourteen days.

Fortunately sympathetic ophthalmitis is a rare disease. Thus, of 108-416 patients seen in private and clinical practice by Mooren, there were 146 cases of sympathetic inflammation, or 1 to 742 (0.134 per cent.). O. Becker, among clinic patients, found 0.15 per cent. of sympathetic ophthalmitis. Accurate statistics as to the frequency with which sympathetic ophthalmitis follows perforating wounds are wanting. That the disease is more frequent in men than in women is to be expected, since men are more exposed to injury. It is asserted that it is more frequent in summer than in winter, owing to the greater intensity of light and the greater virulence of bacteria in the warm season.

Symptoms.—The clinical features of sympathetic ophthalmitis are by no means confined to this disease, since similar changes follow on general diseases and even occur apparently spontaneously (Schirmer). Hence the diagnosis can be made only by taking into consideration the history of the case, the interval between the injury and the involvement of the second eye, and the result of treatment. The process is an inflammation of the uveal tract, and may present either the serous or the plastic form. Where the

pathologic process begins is as yet undetermined; but, clinically, the alterations are found first in the iris. Rarely does the disease manifest itself by changes in the optic nerve.

1. IRITIS (UVEITIS SEROSA).—The mild cases, known as iritis, or uveitis serosa, show slight pericorneal injection, and slight discoloration of the iris. The iris is adherent to the lens-capsule, the pupil is small, and the vision is reduced. These symptoms come on insidiously: *i.e.*, without pain. Dots appear on the posterior surface of the cornea. The anterior chamber is increased in depth, the media are cloudy, and the optic nerve, while generally hazy and red, may show papillitis. The tension is at first slightly increased, then becomes variable, and in the later stages is permanently reduced. From hyperemia the blue iris appears greenish and the dark one looks brownish. As was noted by Pagenstecher many years ago, sympathetic iritis permits enlargement of the pupil by atropin in spite of adhesions. Recession of the iris-periphery is often seen. After a period lasting from a few weeks to several months, the eye, as a rule, recovers completely under proper treatment. The prognosis is favorable. Schirmer states that it is always favorable in the pure serous type of sympathetic uveitis, and he can conceive of the loss of such an eye only through increased intra-ocular tension. The fact must not be overlooked, however, that the case may appear to be of the pure serous type, when, in reality, it is the beginning of the plastic variety. Such cases give an unfavorable result.

2. SYMPATHETIC FIBRINOUS UVEITIS is the commonest as well as the gravest form of sympathetic ophthalmitis. As a rule, the first noticeable symptom is a slight ciliary injection. This may or may not be preceded by diminished accommodative power. Careful examination under a magnifying glass will show the presence of dots in the layer of Descemet, but these are often overlooked, and generally the patient does not consult the surgeon until vision becomes reduced. The eye will then show slight pericorneal injection, muddiness of the aqueous humor, discoloration of the iris, the pupil being slightly contracted and readily enlarging on the use of atropin—a sign that the inflammation is not very acute. The changes in Descemet's layer generally precede the formation of posterior synechiæ. The tension is usually slightly increased, but may be normal. Pain is a noteworthy symptom only in the rare cases running an acute course, and such cases may show swelling of the lids and conjunctival chemosis. Ophthalmoscopic examination not rarely shows inflammatory changes in the papilla, as well as marked vitreous opacities. The frequency of the ophthalmoscopic changes has not been accurately determined, but Schirmer estimates that they exist in the earliest stages of sympathetic ophthalmitis in one-half of the cases. This form of papilloretinitis resembles the chorioidoretinitis of the secondary stage of syphilis. The papilla is abnormally red or grayish red, with blurred edges and without noteworthy swelling. A delicate grayish opacity passes from the disc into the retina. The veins are dark and somewhat tortuous; the arteries are normal. In many in-

stances retinal changes are concealed by diffuse or dust-like opacities in the vitreous. Retinal hemorrhages are rarely associated with these changes. Chorioidal changes have been described by von Graefe, Haab, Hirschberg, and others.

Keratitis punctata and turbidity of the aqueous humor are soon followed by posterior synechiæ. They spring from the pupillary margin of the iris and can be torn loose by strong mydriatics, but they also arise from the periphery, when the pupil is widely dilated. If the synechia is circular, the iris is bulged forward by the hindered flow of aqueous humor, and intra-ocular tension is increased. This occurs only in a few severe cases. The formation of flat adhesions between the iris and lens-capsule is characteristic of typical malignant uveitis, and cannot be prevented by any mydriatic. This fibrinous exudation fills the entire posterior chamber and the space between the ciliary body and the lens. Hence, the iris is pushed forward, and the anterior chamber is shallowed. Soon the masses of exudation shrink, followed by the ominous retraction of the iris, particularly at its periphery, thus showing that the surgeon has to deal with the severest form of sympathetic ophthalmitis. Meanwhile the iris looks macerated, and, owing to the marked swelling, it is disposed in radial folds, which remain because of uneven contraction of the retro-iridal deposit. Large, tortuous vessels are visible, and are due, not to arterial hyperemia, but to swelling from compression of their trunks in the ciliary body. The pupillary area becomes filled with a fibrinous exudate, which gradually increases in thickness and is the cause of the loss of vision. In the early stages the iris is soft and is easily torn, but later it becomes rigid and somewhat elastic. Where the corneal deposits are numerous, they become agglutinated, and, falling to the bottom of the anterior chamber, simulate hypopyon, which disappears later in the course of the disease. This condition was named purulent uveitis by Deutschmann, but Schirmer holds this to be only a severe form of fibrinous uveitis.

In the further course of the disease the cornea loses its transparency, partly because of the increase of intra-ocular tension, partly by reason of cyclitis. Its deepest layers show fine, gray opacities, either diffused over the whole membrane or limited to its central part. The lens, also, becomes intransparent from the development of new vessels. Sympathetic fibrinous uveitis is eminently chronic in its course, lasting for months or years before the turning-point is reached. In the few cases in which the globe is not destroyed a cure may take place, but recurrences of inflammation are so frequent that the eye must have been free of inflammation for a year before the surgeon can regard it as saved. Some of these cases will have useful vision by the clearing up of the opaque media. In others vision can be restored only by the performance of iridectomy, by the extraction of the opaque lens, or, in some few selected cases, by the drilling operation of Tyrrell. These operations are not to be lightly undertaken, and are practicable only when a long period of quiescence has existed. If an iridectomy

is made previous to this stage, the coloboma soon fills with fresh exudation, and the visual result is *nil*. As a rule, sympathetic fibrinous uveitis leaves the eye soft and sightless, with detached retina and opaque media.

3. SYMPATHETIC PAPILLORETINITIS, which is frequently present as an accompaniment of sympathetic uveitis, is in rare instances the sole manifestation of sympathetic ophthalmitis. The papillitis is of moderate degree; the papilla may be slightly more prominent than normally; its outlines are blurred, and its color is either more grayish or more red than under normal conditions. The surrounding retina is somewhat opaque and swollen, and sometimes it shows small hemorrhages. The arteries are either of normal or of increased size; the veins are broadened and tortuous. Visual acuity is usually only moderately reduced. In only 1 of 17 cases recorded by Schirmer was vision reduced to counting fingers at three or four feet. The visual field may be of normal extent, but usually it is contracted. The color-sense is generally normal. The prognosis of sympathetic papilloretinitis is favorable, provided it is not the forerunner of the generalized form of sympathetic ophthalmia. The treatment comprises enucleation of the exciter, when indicated; the local use of atropin; and the internal use of mercurials.

4. OTHER SYMPATHETIC AFFECTIONS have been described, but have not gained recognition among modern ophthalmic writers. Thus, sympathetic optic-nerve atrophy, sympathetic cataract, sympathetic detachment of the retina, and conjunctivitis, keratitis, scleritis, and canities have been recorded. At the present time there exist no good reasons for considering these conditions as sympathetic affections.

Prognosis.—From what has been written it will be apparent that the prognosis of sympathetic fibrinous uveitis—to which class most cases of sympathetic ophthalmitis belong—is exceedingly grave. Randolph, writing in 1898, was able to gather from literature only 19 cases of well-established recoveries. Gumpper, in the same year, in an inaugural dissertation, recorded 65 cures, of which only 25 had been examined one year after the cessation of inflammatory symptoms. Schirmer, who had 5 recoveries in 35 cases, thinks the disease less severe to-day than in former times. Surely prevention of this disease is much more important than therapeusis. As regards the form of sympathetic ocular disease present, the greater the fibrinous exudation and the thicker the iris, the more unfavorable is the prognosis. Uveitis serosa offers a favorable prognosis and the same is true of sympathetic papilloretinitis. The prognosis also is favorable in cases where sympathetic ophthalmitis has developed after removal of the exciter.

Pathology.—While it is not feasible to discuss the pathologic anatomy of sympathetic irritation, that of sympathetic inflammation is of great interest. As is clear, the changes in the exciting eye are much better known than those of the sympathizer, but, as far as our knowledge goes, it can be stated that they are identical in both cases.

The findings in all cases of more chronic, fibrinous uveitis are these:

All of the three portions of the uvea contain disseminated agglomerations of lymphocytes (round cells), which are accompanied, in cases of severe inflammation, by a diffuse infiltration of the whole tissue with the same type of cells. After disappearance of the inflammatory process the characteristic structure of the uvea is lost, and instead a pigmented connective tissue, poorly supplied with blood-vessels, is encountered. Besides this, the iris and the ciliary body are covered with a copious fibrinous exudate, which strongly tends to organization, while the chorioid never produces such an exudate.

Usually the infiltration of the iris is more or less diffuse, while in the ciliary body the most pronounced infiltration occurs in the space between the muscular and the pigmentary layer. In the chorioid the main seat of the process is the layer of the large vessels and in the suprachorioidea, while the inner layers, and especially the capillary layer, are not at all or only slightly affected. It is interesting to state that in the chorioid very often tubercle-like formations with giant cells are found, which, however, have nothing to do with tuberculosis.

Comparatively slight are the changes in the optic nerve, papilla, and retina. In the latter atrophy of the nervous elements and slight increase of the supporting tissue occurs. The papilla often shows an edematous infiltration, but seldom an extensive cellular infiltration, while the latter is often very pronounced in the optic nerve. The episcleral vessels are hyperemic and the tissue around them is loosened and edematous. In the blind end of the subvaginal space of the exciter often a dense infiltration of small round cells is present.

The bacteriologic findings in this disease are of a very contradictory nature. In a number of cases bacteria have been found, most often the staphylococcus pyogenes albus, but in no case has the proof been established that a certain bacterium has been the cause of the sympathetic inflammation. By this, however, it is not meant to say that sympathetic ophthalmitis is not of bacterial origin. Further investigations are necessary.

Pathogenesis.—The limits of this work prohibit an exhaustive account of the various experiments which have been made and the theories which have been advanced to account for the transmission of disease from the exciter to the sympathizer. It is definitely settled that the pathogenesis of this process cannot be determined by experiments made on the lower animals. The old theory of reflex action via the ciliary nerves has been abandoned, and modern ophthalmologists favor the view that sympathetic ophthalmitis is of bacterial origin, but the germ remains to be found. It is supposed that the infection spreads along the sheath of the optic nerve to the chiasma, and thence along the sheath of the other optic nerve. The bacteria theory of sympathetic ophthalmitis cannot be considered as established until the specific organism shall have been identified. One fact which tends to disprove the theory of direct transmission is this: that the infiltration becomes less and less toward the chiasma.

Although we do not know with certainty to-day in what way the healthy eye is involved in inflammatory changes through the disease of the other eye, the theories attempting an explanation are numerous. Leaving aside the theory of sympathetic irritation (transmission of a nervous irritation through the course of the ciliary nerves), only those hypotheses shall be mentioned which deal with inflammation of the sympathizer.

I. NERVE THEORIES.—*1. Optic-Nerve Theory.*—The assumption was that, owing to the well-known crossing of the optic fibres, the inflammatory process, arriving at the chiasma, was from there transmitted along the optic nerve of the other eye. This view, assuming the reflection of an irritation which caused inflammation, according to modern physiologic knowledge appears untenable.

2. Ciliary-Nerve Theory.—This theory supposes that, in the sympathizing eye, an irritative condition of the ciliary nerves is produced which, in a manner as yet unknown, is transmitted to it and causes here a condition of irritation that gradually leads to inflammation. Müller based this theory on merely anatomic reasons. More important are certain clinical observations. Closer consideration, however, shows that this theory cannot be sustained.

II. BACTERIA THEORIES.—With the advent of bacteriology, and with the observation that the majority of ophthalmias are accompanied by bacterial growth, theories have been advanced to explain sympathetic inflammation. The oldest one is that of transmission by metastasis, which was advanced by Berlin. He inferred that the bacteria of the primarily diseased eye obtained access to the general circulation, thus being brought to the uvea of the other eye. Here, finding a congenial soil, they were said to become the cause of the sympathetic disease. Arnold believed that, by a retrograde way through the venous channels, the propagation occurred; while still others, with Leber, thought that the trouble of the sympathizer was accomplished by a continuous growth of the infecting microbe along the lymph-vessels. Schmidt-Rimpler has been a strong advocate of this theory; but it must be remembered that, as yet, in no case scientifically studied has the evidence been established for one or the other mode of propagation. Direct experiments always have had a negative result.

III. COMBINED THEORIES, mainly advanced by E. Meyer and Schmidt-Rimpler, attribute to primary irritation of the ciliary nerves the possibility that pathogenic microbes can find in the tissues innervated by these nerves a suitable soil. Unfortunately very many clinically well-observed facts absolutely cannot be reconciled with this view. For instance, the observation that after an injury to one eye sympathetic inflammation of the other eye never occurs within less than fourteen days. In the same way, this theory leaves the fact unexplained that, after enucleation of one eye, the sympathetic affection of the other can occur as long as many weeks later.

IV. TOXIN THEORIES.—Sympathetic inflammation is not caused by a transmigration of the microbes themselves, but only by that of the products

of their metabolism. What speaks most against this explanation is that, even after extirpation of the primary focus, the inflammation pursues its ordinary course in a great number of cases, while in intoxications the effect of the toxin gradually lessens, as its amount is diminished. This theory affords a satisfactory explanation only for the pure papilloretinitis sympathetica, while it certainly cannot be applied to the ordinary cases.

Prophylaxis.—The prevention of sympathetic ophthalmitis is much more feasible than its cure, and efforts in this direction should begin immediately after the receipt of an injury. The strict aseptic treatment of all wounds should be followed, but unfortunately it will often happen that the wound is infected before the surgeon is consulted.

In cases of injury which cannot remain under the constant observation of an ophthalmic surgeon, the general practitioner should make daily tests of the vision of the good eye, since failure of vision is the one symptom likely to attract the attention of the non-expert.

Prophylactic measures are:—

1. PREVENTIVE ENUCLEATION.—Prophylactic enucleation is indicated whenever there exists a uveitis of bacterial origin which does not yield to proper treatment in reasonable time.

2. PROPHYLACTIC EXENTERATION may take the place of enucleation; but for two reasons (infection of the stump of the optic nerve and the possibility of leaving portions of the uvea behind) this method is not perfectly reliable.

3. PROPHYLACTIC NEUROTOMY (opticociliary) has not proved to be a sufficient protection.

4. RESECTION OF THE OPTIC AND CILIARY NERVES does not furnish a sufficient protection for the other eye.

The normal operation remains, up to date, the enucleation. Exenteration should be restricted to panophthalmitic bulbs, while neurotomy ought to be altogether abandoned.

The most certain of all prophylactic measures is the removal (enucleation) of an injured eye, subject to the rules governing this operation. If the second eye remains well for a period of four weeks after enucleation, the prophylaxis is practically absolute.

Treatment.—The indications for treatment of sympathetic ophthalmitis are (1) removal of the exciter if blind and (2) the use of remedies to control the inflammatory process in the sympathizer.

1. Removal of the exciter, in a case where sympathetic ophthalmitis is fully developed, is not to be considered, if the exciter possesses useful vision. In case there is perception of light, but the eye has remained hypotonic for several weeks, or where corneal opacity is so extensive as to preclude the possibility of the restoration of useful vision, an enucleation of the exciter will be in order. Attempts to improve the condition of the exciter by making broad iridectomies have generally proved unsatisfactory. If tension is increased, it is better treated in these cases by paracentesis of

the cornea or by sclerotomy. If an enucleation of the exciter is not in order, it should be treated with atropin, hot compresses, and possibly subconjunctival injections of bichlorid of mercury (1 to 2000).

2. Treatment of the sympathizer includes local and general measures. Atropin should be used three times a day, and the patient should be kept in a dark-room during the more acute period of his disease.

The intra-ocular injection of antiseptics, as advised by Abadie, is generally condemned by American and German surgeons. The subconjunctival injection of mercuric bichlorid seems to have been beneficial in a few cases.

The internal use of large doses of salicylate of sodium—120 to 180 grains daily in divided doses, fortified by the simultaneous use of brandy—has given good results in the hands of Gifford, who regards it as the most important remedy. Heuse has recorded several cases which were favorably influenced by the daily use of 60 to 75 grains of the same remedy. Aspirin, in 15-grain doses, four or five times a day, may be substituted for salicylate of sodium.

The older surgeons placed their trust in large doses of mercurials given by the mouth or by inunction. Schirmer gives the first place to mercury. Small doses of calomel, frequently repeated, seem to be of value. The local application of heat in the form of poultices, frequently changed, has been advised by Gifford.

Hypodermic injections of pilocarpin sometimes do good and should be tried.

Sympathetic papilloretinitis should be treated locally with atropin, dark glasses, correcting lenses, and the use of hot compresses. Internally mercury or iodid of potassium should be administered. Pilocarpin may be used advantageously. It is inadvisable to keep these cases in a dark-room unless other evidences of sympathetic ophthalmitis are present. In its pure form, sympathetic papilloretinitis offers a favorable prognosis.

Without exception an operation of any kind will be out of order until long after signs of acute inflammation have disappeared. Late in the history of the case an iridectomy may be of some value. If the chief obstacle to vision is an opaque lens, an extraction can be made; or the drilling operation of Tyrrell or the procedure of Critchett can be tried. In Critchett's operation two cataract-needles are used. One holds the lens fixed while the other makes a small tear in the capsule. The points are then separated to enlarge the rent. Some lens-matter escapes and usually there is no reaction. The operation is repeated at intervals of a few weeks until the lens is absorbed. Critchett, Story, and Oliver have practiced this operation with success.

Tyrrell's operation is performed with one needle. It is passed through the cornea near its outer part. It is then made to penetrate the anterior capsule (taking care not to injure the iris) to the depth of one millimetre. The handle is then rotated so that the instrument will act as a drill. The operation can be repeated several times at intervals of a month.

INDICATIONS FOR ENUCLEATION.

Indications for enucleation are furnished either by the condition of the eye or by the state of the orbit.

I. Among the ocular reasons for enucleation, evisceration, or Mules's operation, or one of the modifications of these procedures, are the following (modified from Czermak):—

1. In recent severe wounds, in which the cornea or sclera is lacerated and the intra-ocular contents are partly or wholly lost; wounds which destroy the possibility of restoration of useful vision; wounds with the lodgment of a foreign body which cannot be removed. However, many lacerations of the cornea and some scleral wounds, if not infected, admit of recovery under proper treatment. The prolapsed iris or vitreous is to be cut off, the scleral wound is to be approximated with sutures, the injured lens is to be extracted, etc.

2. In all conditions liable to produce sympathetic ocular disease, the offending eye, if blind, is to be removed. Such conditions include iridocyclitis, iridochorioiditis, with shrinking of the globe; suppurative inflammation following wounds, operations, or the lodgment of foreign bodies; perforating corneal ulcers with iridocyclitis.

3. In cases where sympathetic irritation or inflammation has already appeared in the "good" eye, provided the exciter is blind, or possesses only a limited amount of vision. Here the differences between sympathetic irritation and inflammation are clear-cut: enucleation cures sympathetic irritation, while it has little or no influence over sympathetic ophthalmitis. If the exciter possesses useful vision, and sympathetic ophthalmitis has already developed, the removal of the exciter is not to be considered, since, after the case has run its course, the probabilities are that the exciter will possess the better vision of the two.

4. In uncontrollable hemorrhage from an injured eye, even if the wound is small. This occurs at times in degenerated globes (total ectasia, glaucoma, hydrophthalmos).

5. In spontaneous chronic iridocyclitis with atrophy of the globe; in glaucoma with degeneration of the ocular tunics; in eyes blind from detachment of the retina or luxation of the lens, provided the patient suffers from annoying photopsia or loss of sleep from pain and recurring attacks of inflammation.

6. In general enlargement of the globe through thinning of the sclera (total ectasia of the bulb).

7. In ulceration of the cornea appearing in an eye with total staphyloma or in an eye which has undergone degenerative glaucoma. These conditions lead to rupture, hemorrhages, panophthalmitis, or insidious iridocyclitis.

8. In all malignant intra-ocular tumors where complete extirpation cannot be performed. This concerns all tumors of the retina, ciliary body,

and chorioid, and those tumors of the iris which involve the margin of the ciliary body. Small tumors of the iris, so situated that they can be entirely removed, are to be subjected to iridectomy.

9. In all epibulbar malignant tumors whose attachment to the globe is so close as to render removal of all the growth incompatible with the integrity of the eye, or tumors in which the orbital tissues are also involved.

10. In primary conglobate tuberculosis (granuloma) of the iris and chorioid.

11. In all cases of phthisis bulbi where there is shrinking pain on pressure.

12. In cases where other operations (opticociliary neurectomy, excision of the cervical sympathetic for relief of pain in glaucoma) have failed to relieve suffering, the eye being blind.

13. In panophthalmitis.

II. The orbital indications for enucleation are:—

1. Inoperable tumors encroaching on the walls of the orbit, producing exophthalmos and lagophthalmos, and leading to destruction of the globe by painful suppurative inflammation.

2. In orbital cellulitis, if the eye is already lost and the surgeon believes that life will probably be saved by the operation.

ENUCLEATION AND ITS SUBSTITUTES.

Removal of the eyeball, either complete (as in enucleation) or partial (as in Mules's operation, Hall's operation, or their modifications), is to be done under general anesthesia, unless the condition of the patient is such that the inhalation of chloroform or ether will be dangerous. In old alcoholics, for example, general anesthesia is dangerous and may be replaced by the local use of cocain or holocain, aided by the use of adrenalin or suprarenal solution. These remedies can be dropped on to the conjunctiva, and, after this membrane is incised, further use can be made of the solutions before the next step is taken. Some surgeons prefer to inject the solution into the orbital tissues by an hypodermic syringe. This method may cause great depression, cold perspiration, or syncope, while dropping cocain in the wound, as the operation proceeds, is tedious and often unsatisfactory. In this manner the parts can be sufficiently anesthetized, there being little or no pain, except at the moment of cutting the ciliary nerves surrounding the optic nerve. In case of evisceration (Mules's operation) the pain is practically *nil* under holocain or cocain. Before enucleation the conjunctiva and skin of the lids and adjacent parts are to be thoroughly cleansed with soap, water, and bichlorid solution. After the globe has been removed, it is well to flush the orbit with hot sterile water or with bichlorid solution.

Technique of Enucleation.—Of the various methods of enucleation two are in common use, viz.: Bonnet's method and Arlt's modification.

Bonnet's Method.—The object of this operation is the removal of the eyeball without injury to Tenon's capsule. The instruments required are a speculum, tissue-forceps, fixation-forceps, scissors, strabismus-hook, curved enucleation scissors, needles, suture material, and needle-holder. The speculum having been introduced, and the eyeball being held by an assistant, the surgeon rapidly separates the conjunctiva from around the cornea, and dissects it back as far as the insertions of the recti muscles. The tendons are lifted up, one at a time, by the strabismus-hook, and are cut off close to their insertions. The subconjunctival tissue is likewise lifted up by the hook. The next step is to dislocate the globe forward by pressing the speculum backward, this being done so as to facilitate the cutting of the optic nerve. This is accomplished by means of long, curved scissors, which are held closed and are passed backward until the nerve is felt as a tense cord. The blades are then opened, the nerve engaged, and cut close to the sclera. The globe is then held in the left hand and the oblique muscles are severed, together with the ciliary vessels, nerves, and any adhering tags of tissue. The operation is completed by the use of a purse-string suture, passed

Fig. 354.—Enucleation scissors.

through the conjunctiva and through each of the recti tendons. Some operators, after cutting all other muscle attachments close to the sclera, leave a piece of the tendon of the external rectus muscle attached to the globe, and hold it with fixation-forceps to steady the eye while cutting the optic nerve. The dusting of iodoform or boric acid into the conjunctival sac and the pouch, left by removal of the eye, is unnecessary.

In cases requiring resection of the optic nerve back to the foramen opticum, Bonnet's method is somewhat modified. After removal of the globe, as described above, the surgeon feels for the stump of the optic nerve with his right index finger, and grasps the nerve with fixation-forceps held in the left hand. After seizing the nerve-stump he cuts it off at the apex of the orbit by means of curved, blunt-pointed scissors.

Arlt's Method.—This operation requires a speculum, toothed forceps, and scissors. The tendon of the internal rectus muscle is seized with the overlying conjunctiva and the grasp is retained until the end of the operation. The muscle is cut external to the forceps. Through this opening one blade of the scissors is passed under the inferior and superior recti tendons, which are severed in succession. Then the optic nerve is cut, the

globe being drawn forward and turned outward to facilitate the section. Last, the external rectus and the two oblique muscles are severed close to the globe and the operation is finished. The purse-string suture may be used or not, as the operator desires. The author favors its use, believing that the conjunctival suture aids primary union and often prevents the formation of a button of "proud flesh." It is well not to draw the suture too tightly, since blood may accumulate behind it and cause great pain. Many operators place a small piece of gauze in the orbit before tying the suture. This gauze is changed daily for a week. The dressing used is dry gauze and a bandage.

Arlt's method is exceedingly rapid, but it is not suited to eyes whose walls are weak, since the pressure used in the operation will cause rupture of the globe and escape of the contents, increasing the danger of infection.

Among the accidents, mistakes, and sequelæ of enucleation are the following:—

1. Removal of the Good Eye has occurred in at least one instance of which the author has received reliable information. In a case in which the pathologic condition does not show externally—as, for example, an intraocular tumor—it will be advisable for the surgeon to make a mark on the lid of the eye to be removed.

2. Hemorrhage, usually insignificant after enucleation, has led to death in a few instances in bleeders and old alcoholics. Usually pressure by the finger at the apex of the orbit will stop it, and the compress bandage will prevent its recurrence.

3. Inability to Deliver the Globe may occur because of smallness of the palpebral fissure, which, of course, must then be enlarged; but usually the surgeon will find that the optic nerve has not been severed.

4. Extension of Disease beyond the Globe is often encountered in malignant growths which have perforated the eye posteriorly and have involved the optic nerve and adjacent tissues. Here the optic nerve must be resected back to the foramen opticum, and all infected tissue should be removed from the orbit.

5. Perforation of the Globe may occur even in the hands of a careful surgeon, if the sclera is unusually thin. The operation is to be finished, all escaping liquids being carefully wiped away and the orbit thoroughly flushed with bichlorid solution. If the globe collapses, the operation is to be finished by careful dissection, the eye being held with a sharp hook. Such an accident is not uncommon after perforating wounds, ulcers, or tumors.

6. Panophthalmitis with Orbital Thrombosis, producing a hard, lardaceous condition of the orbital tissues, may confuse the operator. Here the surgeon has simply to follow the outline of the thickened sclera to remove the globe.

7. Occipital Pain and Ecchymosis of the Lids are sometimes seen after the most careful enucleation. The pain stops in a few hours and the ecchymosis disappears in ten days.

8. Orbital and Palpebral Abscess, following enucleation, are rare complications. They are to be treated on general surgical principles.

9. Fatal Meningitis after enucleation is of rare occurrence. The author has never seen a case, although he does not hesitate to enucleate panophthalmitic eyes. It is due to infection either by extension of germs of panophthalmitis to the sheath of the optic nerve, and thence to the brain; or to unclean instruments, sutures, solutions, gauze, or fingers; or to contact with pus from the eye by rupture of the globe during the enucleation, or from pus derived from the tear-sac. Von Graefe, who lived before the era of aseptic surgery, observed meningitis twice after enucleation of panophthalmitic eyes, and advised against the operation. Most modern ophthalmologists, however, do not hesitate to enucleate them, thus greatly relieving pain and suffering and shortening the period of treatment. Some surgeons, however, think best to eviscerate the globe in panophthalmitis. If an orbital phlegmon exists with meningitis, Noyes advises that deep incisions be made to let out the pus, and deep irrigation with strong antiseptics is to be used. If meningitis occurs after enucleation, it is not necessarily fatal, although the condition is very serious.

In 1896 a committee of the Ophthalmological Society of the United Kingdom investigated this subject. Of 10,734 cases of simple excision, in 7 fatal meningitis ensued; in all of these the eye which was removed was suffering from suppurative panophthalmitis. It is not certain that the operation caused the meningitis, since cases of panophthalmitis have been recorded in which the eye was not removed and meningitis has occurred. One case of fatal meningitis has followed evisceration. Since this operation has been much less frequently performed than enucleation, it is an open question which operation is freer from risk. The committee could learn of no case of meningitis following Mules's operation or the insertion of an artificial globe into Tenon's capsule. The author has had one death during enucleation under chloroform anesthesia which was due to the fact that an arrow-shaped piece of steel had pierced the globe, had passed into the sphenoidal fissure, and had injured the brain.

Technique of Evisceration.—The instruments required are speculum, fixation-forceps, scalpel, scissors, scoop, suture material, needles, and needle-holder. The speculum being in place and fixation-forceps holding the globe, the scalpel is used to puncture the sclera at a point five millimetres posterior to the corneoscleral junction. Into this opening one blade of the scissors is introduced, and a cut is made concentric with the cornea. Thus, the anterior segment of the globe is removed. The iris, ciliary body, chorioid, lens, and vitreous humor are removed with the scoop or spoon. Tags of chorioid and retina will adhere to the venæ vorticosæ and nerve-head. Such adherent pieces can best be removed by wiping with gauze held in forceps, which are rotated rapidly within the globe. The circular wound is now altered by removing small triangular pieces of sclera at the insertions of the external and internal recti muscles. The cavity is irrigated with sterile water or

with a bichlorid solution, and the wound is closed by five or six sutures passed through conjunctiva and sclera. A gauze dressing and compress bandage are applied. Healing is usually uneventful, but Knapp has reported a case of evisceration followed by thrombosis of the vorticose and orbital veins. There is frequently marked chemosis following this operation. The sutures are removed on the seventh or eighth day.

Evisceration with Insertion of an Artificial Vitreous (Mules's Operation).—This operation is simply an evisceration plus the placing of a ball of glass or metal (gold) within the scleral cup. To facilitate the introduction of the artificial vitreous, the introducer of Mules or that of Todd is used (Fig. 355). The globe should not be so large as to stretch the sclera when sutures are introduced. Two sets of sutures are to be used: the scleral ones are directed vertically and when tied present a horizontal scleral wound, while those through the conjunctiva are passed horizontally and when tied the conjunctival wound is vertical. Catgut is used for sewing the sclera, and silk for the conjunctiva. A pad of gauze and a firm compress bandage

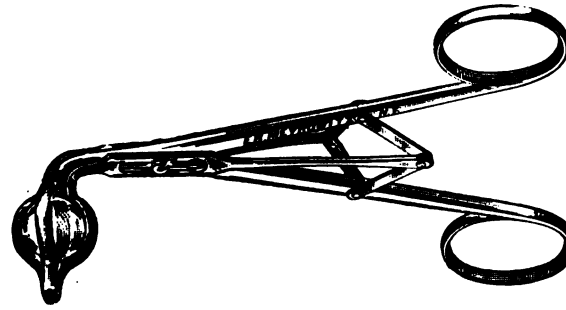


Fig. 355.—Artificial-vitreous introducer. (Todd.)

are applied and reapplied during the period of swelling. There is always considerable swelling and some pain for several days after this operation. The success of Mules's procedure demands rigid attention to asepsis. The cosmetic results are beautiful, since the rotations of the artificial eye, placed on the stump, exceed those obtained after enucleation. Furthermore, the natural contour of the lids is retained, and there is an absence of the annoying conjunctival secretion which follows the wearing of a shell after enucleation. In the author's cases the artificial eye was inserted two or three weeks after operation. The globe is extruded in from 12 to 15 per cent. of the cases. Once the wound is firmly closed, it does not, as a rule, subsequently reopen, but it has been known to do so as late as seven years after the insertion. No case is recorded in which the glass globe has broken in the sclera. Silver globes have sometimes produced argyrosis of the stump.

Sclero-optic Neurectomy (Hall's Operation).—This operation, which originated with Dr. Ernest Hall, of Victoria, B. C., consists of an evisceration to which is added an excision of the posterior part of the scleral cup and resection of the optic nerve. After cutting away the sclera to an extent

sufficient to include the ciliary body and eviscerating the globe, the speculum is inserted within the ball, thus holding both lids and edges of the sclera open. "The point of entrance of the optic nerve is then grasped with two forceps and the scissors inserted as close to the nerve as is possible to avoid wounding of the ciliary arteries, and a circular incision is made in the sclerotic, freeing the optic nerve, which is then drawn forward and severed about ten millimetres from the sclerotic junction, thus removing a section of the optic nerve. The sclerotic and conjunctiva are closed vertically to give normal tension to the internal and external recti, as lateral movement is of greater importance than vertical. After the artificial eye is adapted there is perfect movement within thirty-five degrees laterally and twenty degrees vertically." This operation has been modified by Huizinga, who removes a larger section of the posterior scleral cup, cuts and removes the optic and ciliary nerves, and inserts an artificial vitreous. He has named this operation *eviscero-neurotomy.*

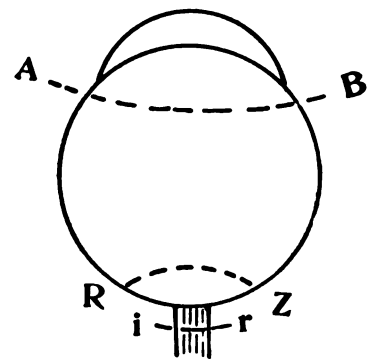


Fig. 356.—Sclero-optic neurectomy.

The tissues anterior to the curved line, *A-B*, are to be removed. *R-Z*, Incision in posterior part of the sclera. *i-r*, Part of the optic nerve to be excised.

Implantation of a Sphere into Tenon's Capsule (Adams Frost's Operation).—This operation was done by Frost in 1886, as follows: "The conjunctiva having been divided in the usual way, each rectus muscle in succession was seized with forceps, divided, and secured by a ligature passed through it and through the conjunctiva; the enucleation was then completed, the four recti being held apart by means of the sutures. Mules's sphere was introduced into the capsule of Tenon without any difficulty; opposite recti, with the conjunctiva, were then united across it by passing one of the ligatures through the opposite tendon, the other ligature being then removed." This operation has been practiced by Lang, who unites Tenon's capsule horizontally by three silk sutures and then sews the conjunctiva separately. Other modifications have been made by Morton, of Minneapolis, and Oliver, of Philadelphia. The sphere can be made of glass, celluloid, silver, or gold. Fenestrated aluminum spheres were proposed by Bryant, of Omaha.

Immunity to Sympathetic Ophthalmitis.—The comparative value of these operations as regards immunity to sympathetic ophthalmitis was investigated in 1896 by a committee of the Ophthalmological Society of the United Kingdom. The committee found no case of sympathetic inflammation following simple evisceration (von Graefe's operation), but gives details of five cases after Mules's operation. In all of these, however, the eyes had received injuries likely to produce the sympathetic disease, which is probably to be attributed more to the injuries than to the operations. The committee found no evidence to show that the insertion of an artificial globe into Tenon's capsule lessens the immunity which excision confers. Cases are detailed of sympathetic ophthalmitis following opticociliary neurectomy and neurotomy. Since these operations are practiced to a limited extent, it is inferred that they are not so valuable as enucleation in preventing sympathetic ophthalmitis. Abscission affords less immunity than enucleation.

As regards sclero-optic neurectomy, Hall claims the following advantages: "1. The volume of the pad due to the preservation of a considerable amount of tissue. 2. The extensive movement of the pad owing to the muscular attachments being left undisturbed. 3. The slight risk of paralyzing the muscles during the operation, through injury to the third and sixth nerves. 4. The absence of any tendency to sympathetic disease of remaining eye, on account of removal of the ciliary region in front, and the sclero-neural connection behind."

The Advantages of Mules's Operation versus Enucleation have been presented by Bickerton as follows:—

Enucleation.	Mules's Operation.
1. Complete removal of the globe and its contents.	1. Retention of the frameworks of the eye.
2. No stump; therefore sunken eye.	2. A firm, round globe forming perfect support for an artificial eye.
3. Disturbance of all muscular relations and arrest of movement.	3. Perfect harmony of muscular movements retained.
4. A fixed, staring eye attracting attention.	4. Fitted with a selected eye it defies detection.
5. Patient shuns society.	5. No qualms as to personal appearance.
6. Arrested development of orbit in case of children.	6. No interference with growth of orbit.

OCULAR PROSTHESIS.

The insertion of an artificial eye to cover the defect caused by atrophy or deformity of the globe, or after an operation of the eyeball, is called ocular prosthesis.

After an enucleation or one of its substitutes has been made, it is advisable that the patient should wear an artificial eye. This should be inserted, and worn daily, as soon as the tissues will tolerate its presence. If an enucleation has been made, there is considerable sinking of the soft parts at

the base of the orbit. To obtain the best cosmetic result, under these circumstances, the artificial eye should be of considerable thickness and should be fitted accurately to the soft parts. Formerly, when artificial eyes of a shell form were worn, the cosmetic results of an enucleation were often unsatisfactory. Since the advent of the thick, or "reform," eyes the results have been much more satisfactory. After a Mules operation only the shell form of artificial eye is necessary.

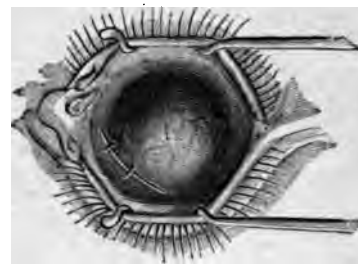


Fig. 357.—Sphere in position. (Fox.)

To insert an artificial eye the patient should raise the upper lid, resting the hand flat upon the forehead; then the eye is pushed beneath the upper lid and, while held in this position, the lower lid is pulled downward, thus admitting the eye to the socket. Insertion of the eye will be facilitated by smearing it with white vaselin. *To remove an artificial eye* the lower lid is to be pulled downward; then the head of a pin or the small end of a bodkin is passed beneath the lower edge of the eye; gentle traction will now lift the eye from its bed and it can be caught in the hand.

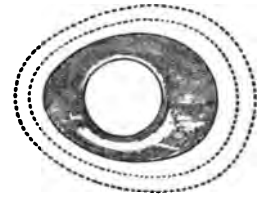


Fig. 358.—Conformer. (Fox.)

The dotted lines show the different sizes of conformer.

The artificial eye should be removed before retiring. It should be wiped dry, but should not be placed in water, since sudden changes in temperature will produce minute fissures. The life of an eye is about two years. When it becomes eroded it should be superseded by a new one. The best eyes are made of glass. Celluloid eyes are cheaper, but are not satisfactory. If the socket becomes irritated, the eye should not be worn for a day or two and antiseptic washes should be used. It is necessary that the eye shall fit properly. Improperly fitted eyes may cause sympathetic irritation or inflammation.

Operation for the Better Support of an Artificial Eye.—Fox has devised the following operation for the better support of an artificial eye in cases where an enucleation has been made at some previous time: An incision, slightly less in extent than the diameter of the gold ball to be inserted, is made through the conjunctiva and tissues of the orbit. The upper lid of the conjunctiva is raised, and, with sharp-pointed curved scissors, the conjunctiva and such connective tissues as lie close to it are dissected off in all directions around the incision, making a pouch into which the glass ball will fit. The edges of the conjunctiva are brought together over the ball and are held by five or six stitches. The sphere is retained in position by a shell, or "conformer," which is left in place for twenty-four hours. In a few days the artificial eye is fitted and can be worn with comfort. Such objectionable features as retained secretion, immobility, and sunken appearance are absent.

Injection of Paraffin after Enucleation has been practiced by Ramsay, of Glasgow. The enucleation is made as in the ordinary manner except that each rectus tendon is sutured to the conjunctiva before the muscle is severed from the eyeball. After removal of the eye the capsule of Tenon is packed with gauze while a purse-string suture is inserted into the conjunctiva. The gauze is then removed and, while the recti muscles are put on the stretch, melted paraffin is injected into the space formerly occupied by the globe. The muscles are united by catgut sutures, the conjunctival suture is tied, and a bandage is applied. The result is a movable stump on which the artificial eye can be fitted. Ramsay reports 4 failures in 22 cases.

Operations for Prosthesis in Cicatricial Orbit.—Occasionally cases occur in which, owing to lime burns, trachoma, etc., cicatricial bands prevent the insertion and wearing of an artificial eye. Operations for the relief of this condition are numerous and most of them are unsatisfactory. Incisions do no permanent good. Lining of the enlarged cavity with half-skin flaps (Thiersch flaps cut very thick) has given fair results in Gifford's hands.

CHAPTER XIX.

DISEASES OF THE ORBIT.

By WILLIAM T. SHOEMAKER, M.D., of Philadelphia,

Assistant Ophthalmologist and a Chief of Clinic to the German Hospital; Dispensary Ophthalmic Surgeon to the Presbyterian Hospital; Oculist to the Pennsylvania Institution for the Deaf and Dumb; Associate Member of the American Ophthalmologic Society.

THE orbit is subject to congenital anomalies, tumors, inflammations, and injuries.

CONGENITAL ANOMALIES.

Congenital anomalies of the orbit are found as various degrees of alteration in the size and shape of this cavity, with a corresponding influence upon

Fig. 359.—Cyclops. (After VAN DUYSE.)

the orbital contents. The defect may be unilateral or bilateral; or, falling within the field of teratology, all degrees of anterior dichotomy or of fusion may result in the formation of one, three, or four orbits.

A two-headed monster would, of course, have four orbits. A double-faced monster may have four or three according to the degree of duplication.

Cyclopia is a condition in which there is but one orbit and a single eyeball. The orbit is usually large and quadrangular, and is situated at the root of the nose. Immediately above is generally a proboscis. The eyeball may be perfectly single or may show degrees of doubling. The older theory for this condition is that of Geoffroy St. Hilaire, who believed that the rudiments of two orbits at first distinct had fused in the process of

development. Hirst and Piersol believe it to be an arrest of development of the anterior cerebral vesicle. According to these authors, there is normally at first material for but one eye, which is later differentiated into two.

The superior wall is sometimes almost vertical, the orbit then being abnormally shallow, the eyeball proptosed, and its movements much limited. An acquired anomaly, of the size and shape of the orbit, of this character is found in those cases in which the eyeball has been removed or destroyed in infancy or in early childhood. As the orbit develops *pari passu* with and in proportion to the development of its contents, a loss of the latter is accompanied by a cessation of the former. The orbit remains small, tends to become horizontally slit-like, and the face develops asymmetrically.

The Orbital Contents may be congenitally defective or deficient. The eyeball may be abnormally small—a condition known as *microphthalmos;* or it may be entirely absent, the anomaly then being *anophthalmos.*

Fig. 360.—Anophthalmos. (Author.)

Microphthalmic eyes may be small, but otherwise normal; or they may be cystic, one or more cysts being present as the result of imperfect closure of the fetal fissure. These cysts are therefore always situated below, and may extend for some distance, even into the lower lid.

Anophthalmos.—About one hundred true cases of congenital absence of one or both eyes have been reported. While the condition is recognized clinically, in a strict pathologic sense it rarely, if ever, exists. Cases of this anomaly which have been subjected to anatomic examination have generally shown the presence of rudimentary eyes. Ten cases are on record in which careful dissection failed to show any trace of an eyeball. The condition is often associated with the presence of an orbito-palpebral cyst. In cases described clinically as unilateral anophthalmos the eye which is present may be normal, microphthalmic, nystagmatic, highly hypermetropic, or colobomatous in its iris and chorioid. The lids are generally well formed, but may be small and adherent at their margins. The orbits are often smaller

than normal. The lacrimal glands are generally present. The puncta and canaliculi may be absent. Frequently the rudimentary eye can be felt as a small mass deeply placed in the orbit. Von Hippel has collected 87 cases of anophthalmos: *i.e.*, absence of one or both eyes without discernible cyst-formation or other evidences of a rudimentary eyeball. Of these, 64 were bilateral and 23 were unilateral.

The Ocular Muscles may be anomalous in origin, insertion, or in structure. One or more may be entirely absent. A corresponding loss of function will then exist.

Congenital Cysts of the Orbit are generally dermoid in character, due to a fetal invagination of epithelial-producing cells. They lie against the orbital periosteum, often in a little depression which they have made for themselves. While the most common location is perhaps the nasal side, they have been found in all situations, with greater frequency, however, in and out. They are smooth, have no very fixed attachments, and upon palpation their cystic nature can usually be made out. Before operating for their removal it is very important to diagnose them differentially from

Meningocele and Encephalocele, which may be present in the orbit. These two conditions are also congenital. The first is a prolapse, or hernia, of the meninges with cerebro-spinal fluid contents, projecting through a breach in the retaining skull-wall. The second is the same with the addition of brain-substance prolapsing within the meningeal sac. The bony defect is in the line of a suture. It generally involves the junction of the ethmoid and the frontal bone, the tumor then presenting at the upper, inner orbital angle. Very rarely a posterior orbital meningocele or encephalocele presents near the apex of the orbit. Its diagnosis without exploratory operation is impossible. The following points must be observed: Meningocele and encephalocele are often reducible, and after reduction the bony margin of the aperture through which they came can be felt. Pulsation and hemic sounds can be elicited. The size and tension of these protrusions are variable, depending upon varying blood-pressure within the cranium. A dermoid cyst will present none of these phenomena. A meningocele may become constricted and its neck obliterated, leaving a complete cyst with cerebral fluid contents within the orbit.

Treatment.—Non-interference must be observed in the treatment of meningocele and encephalocele. The treatment of orbital cysts is referred to on page 627.

TUMORS OF THE ORBIT.

Modern pathology has restricted the term "tumor" within narrower limits than have been generally recognized by authors of text-books on ophthalmology. Senn defines a tumor as a localized increase of tissue, the product of tissue-proliferation of embryonic cells, of congenital or post-natal origin, produced independently of microbic causes. This definition

excludes inflammatory swellings. It also excludes cysts, which are the abnormal dilations of pre-existing tubes or cavities. Dermoid cysts and dermoid tumors are to be classified as teratomata, or aggregations composed of various tissues, organs, or systems of organs which do not normally exist where found. The sharp line drawn is of importance, for, while an inflammatory swelling may cause many or all of the clinical symptoms produced by a tumor, the first may be amenable to internal medication. It is said that no authentic case of a neoplasm being so influenced is on record. This section will comprise only the true tumors.

In a case of orbital tumor it is desirable to determine, first, the origin of the tumor or that tissue primarily affected; second, the nature of the growth, regarding its malignancy or its innocence; and, third, the extent to which the adjacent structures have become involved, either mechanically or by infiltration.

Etiology and Location.—Tumors originating primarily in the orbit are comparatively rare, but invasion of the orbit by neoplasms originating in the sinuses and cavities of the head markedly increases the percentage of orbital disease of this character. C. O. Weber, in an analysis of 1013 cases of tumor, found 41 affecting the eyes and orbit, 217 affecting the organs of the mouth and the maxillary bones, and 56 affecting the nose, pharynx, and antrum of Highmore. A certain number of these extra-orbital tumors necessarily found their way into the orbit. Billroth has reported 18 orbital tumors in a series of 217 growths occurring in the region of the head.

In the last few years the use of the Roentgen ray has done much for the more ready determination of the extent and origin of some tumors, especially those of the bones. A sciagraphic comparison of the two sides of the head will often show an increase of the size, and alteration in the shape, of the individual bones affected. A tumor pushing into the orbit will displace the eyeball in front of it. The direction of the displacement of the globe is therefore of diagnostic value. Displacement directly forward, due to a tumor, is indicative of a location within the muscle-cone (Berry). *Mobility in toto,* if elicited, is evidence presumptive that the tumor is not of the orbital wall. *Pulsation,* when present, is an important symptom, and shows that we have a vascular growth to deal with. It is often difficult definitely to localize the pulsation within the tumor, and care must be taken not to mistake for tumor an aneurismal disturbance within the orbit accompanied by exophthalmos. A vascular pulsating tumor may be reduced in size, but not completely obliterated, by pressure and temporary obstruction of the circulation. An aneurism, by similar procedures, will suffer a much greater reduction in size.

Innocence or Malignancy.—Of greatest importance to the patient is the question of malignancy. It is an accepted fact that, the more rapid the growth of a tumor, the more malignant it is. The clinical features of benign tumors are: they grow slowly; they are encapsulated, and because of this they are more or less mobile. They do not infiltrate, do not infect

the lymph-glands, do not recur after complete removal, do not give rise to metastasis, and they endanger life only indirectly. On the other hand, malignant tumors grow rapidly; they have no limitation, but infiltrate surrounding tissues; and, because of this unbounded infiltration, infection takes place, which may be local or general. They are immobile independently of the surrounding infiltrated tissues. They recur after removal, and inherently tend to destroy life. Metastasis belongs to malignant tumors, and shows the tumor to be no longer a purely local condition as it was at first, but that the disease has become general owing to infection. Sutton calls attention to the facts that innocent tumors are apt to be multiple; that sarcoma occurring in a paired organ is likely to arise concurrently in the fellow-organ; and that the occurrence of two primary carcinomata in the same individual is exceedingly rare.

Symptoms.—The symptoms of orbital tumor are those due to pressure and destruction previously described. Pain may or may not be severe, depending upon the amount of pressure, direct or indirect, upon the sensory nerves. Diplopia is often the first indication of the growth, and should always be regarded with suspicion when its cause is not apparent. Late blindness is to be expected in probably the majority of orbital tumors. Early blindness is suggestive of primary involvement of the optic nerve or the parts around the optic foramen.

Prognosis and Treatment.—From what has been said it will be seen that the diagnosis of an orbital tumor as to kind, location, and extent determines the prognosis. The latter contemplates, first, the life of the patient; and, second, the preservation of vision.

General surgical principles must be the guide for treatment. Complete removal by operation must, as a rule, be attempted where possible. Accepting the modern view that every primary malignant tumor is at first a purely local affection, the sooner removal is accomplished, the better. Furthermore, a benign tumor may destroy sight quite as readily as one which is malignant; it is well, therefore, to minimize by early operation the danger of irreparable damage from pressure.

Senn places the average duration of life of persons suffering from malignant tumors of all kinds, without surgical interference, at about three years. Bearing this in mind, certain cases having also other inherently fatal diseases might advantageously be spared an operation.

All tumors must be completely removed with a view to preventing a probable return. Removal of the tumor alone, when possible, will suffice for those which are benign. Malignant tumors are more safely removed with all of the orbital contents. The best results claimed by general surgeons for operation for malignant tumors vary from 15 to 25 per cent. of permanent recoveries.

Tumors situated deep in the orbit may sometimes be removed by an osteoplastic operation without undue destruction of the remaining orbital structures, after the method of Krönlein, which will be described later.

Varieties of Tumors.—The individual tumors which are found in the orbit may be divided into (1) those composed of connective-tissue elements and (2) those composed of epithelial elements. The connective-tissue tumors are lipoma, chondroma, osteoma, sarcoma, neuroma, and angioma. The epithelial tumors are papilloma (cutaneous horns), adenoma, carcinoma (cancer), and squamous carcinoma (epithelioma).

Lipoma.—The general characteristics of lipomata are that they are encapsulated, frequently lobulated, and sometimes congenital. They grow slowly and are benign. Billroth found that in a number of instances they are symmetrical, and like other benign tumors, as noted by Sutton, are multiple. Certain varieties (lipomata telangiectodes) are very vascular and some contain blood-channels (lipomata cavernosa). Although the orbit contains a large amount of fat, lipomata arising actually within the orbit are exceedingly rare. In considering lipoma it should be remembered that in Basedow's disease the orbital fatty tissue sometimes undergoes a general hypertrophy. Such an hypertrophy may also be secondary to an angioma, as was the condition in a case of angioma lipomatodes reported by Knapp.

Angioma.—An angioma is a tumor composed entirely of newly formed blood-vessels. Dilations, enlargements, and communication of pre-existing blood-vessels must be excluded, as are all aneurisms or varicose veins. Depending upon the character of blood-vessels of which they are composed, there are three varieties of angioma, viz.: capillary, cavernous, and plexiform.

Capillary Angiomata, simple nevi, or mothers' marks, are always congenital, and show a predilection for the skin of the face around the orbit. They differ in color, depending upon whether the blood-vessels are superficially or deeply situated (Billroth). The consistence is determined by the relative amount of connective tissue present. Those angiomata having but little connective tissue are soft and easily compressible, while those rich in connective tissue are quite firm and resistant.

Cavernous Angiomata contain irregular communicating blood-spaces. They occur anywhere within the orbit, a favorite site being behind the eyeball, within the muscle-cone. They are larger and more dangerous than the capillary variety, and have a tendency to progress. They may be congenital or may occur at any time during life.

Plexiform Angiomata consist of a number of tortuous blood-vessels which are parallel one with another. They may be composed of arteries, veins, or both. Pulsation is generally present. If the tumor rests against the orbital wall, the underlying bone may be absorbed.

Diagnosis.—All blood-tumors are more or less reducible upon pressure. Bruit and pulsation are generally present in cavernous and plexiform angiomata. These two varieties are also increased in size by all actions which tend to raise the blood-pressure, such as crying, laughing, straining, etc. The capillary angiomata around the orbit are generally found on the lids or near the orbital margin, and are readily recognized. The differential

diagnosis between a true angioma and an aneurismal condition deep in the orbit may be impossible.

PROGNOSIS.—Angiomata, as a rule, are benign tumors. They may, however, be transformed into sarcomata of a most malignant type. They frequently enter into combination with other tumors, supplying the vascular elements and making the condition more serious than were either tumor alone present. Such combinations are angiolipoma, angiofibroma, angioadenoma, angiosarcoma, and angiocarcinoma. They may occasionally dis-

Fig. 361.—Fibrosarcoma of the orbit. (WILSON.)

appear spontaneously after obliterative inflammation. On the other hand, inflammation of a cavernous or plexiform angioma may give rise to septic thrombophlebitis and death from pyemia. Pressure of an orbital angioma might, of course, readily destroy vision, and an erosion of bone might result from a pulsating tumor.

TREATMENT.—If it can be done, excision is the treatment *par excellence* for angiomata. This, in deep-seated orbital angiomata, is often impossible without an amount of destruction which might be considered scarcely justifiable in dealing with a tumor the natural tendency of which is innocent.

Such means as will cause coagulation of the blood and obliteration of the tumor are to be considered. With these comes the great danger of septic thrombophlebitis. As coagulating injections should never be employed without ligature, their use in the treatment of orbital angiomata is to be condemned. Repeated electrolysis is of value in the treatment of superficial angiomata, and might be successfully tried in certain of the more deeply situated ones. Ignipuncture, or puncture with a red-hot Paquelin cautery-needle, is excellent treatment for superficial angiomata which are readily accessible to the needle.

Ligation of the arteries supplying the tumor, or of the main trunks going to the part of the body containing the tumor, has not proven satisfactory. The collateral circulation soon causes a return of the growth. In

Fig. 362.—Orbital tumor in a case of sarcomatosis. (W. T. Shoemaker.)

excising an angioma care should be taken not to cut into the tumor, as troublesome hemorrhage is apt to follow such a procedure.

Lymphangioma is a tumor composed of lymphatic vessels. It is generally congenital, and tends to grow after birth. It contains more connective tissue than does an angioma, and is consequently more firm. Clinically it presents many of the characteristics of angioma. Its occurrence in the orbit is very rare. The prognosis is not favorable.

Chondroma, or Enchondroma, is a tumor composed of hyalin cartilage. It is one of the most benign tumors, grows very slowly, and is so perfectly encapsulated that it is, as a rule, easily removed surgically. Its occurrence in the orbit is very rare, in which case it may be found in the orbital cellular tissue, or with greater frequency in the lacrimal gland. Clinically, it is hard and firm, and may be mistaken for osteoma. If, as enchondromata sometimes do, the tumor should undergo mucoid degeneration, a point of

differential diagnosis between it and an osteoma would be the fluctuation in the degenerated mass. An enchondroma is usually unaccompanied by pain. It is a tumor of childhood or of early adult life.

Sarcoma.—Several varieties of sarcoma may occupy the orbit, such as fibrosarcoma, myxosarcoma, cystosarcoma, osteosarcoma, cylindroma, plexiform sarcoma, and melanosarcoma. The individual embryonal connective-tissue cells may be round or spindle shaped. Of all tumors occurring primarily in the orbit, sarcomata are the most frequent, the most malignant, and the most certain eventually to destroy life. Being without capsules, they rapidly infiltrate, and cause more or less immobility of the eyeball. They are of rapid growth, often attaining great size, and give rise to pain. They are firm, rather uneven of surface, and with few exceptions they are free from pulsation and fluctuation. As a rule, the eyelids remain soft and free. They tend to invade any or all of the surrounding cavities. More often they are intra-orbital secondarily rather than primarily. A primary sarcoma of the chorioid frequently breaks through the sclera into the orbital cavity. Rarely is the eyeball attacked secondarily by a sarcoma originating within the orbit proper.

Although certain varieties of sarcomata are more rapidly destructive than others, notably the cylindroma and the melanotic variety, they are all sufficiently so to allow of no temporizing when once the diagnosis has been made, providing the case is seen early. The only possible chance of removal without recurrence is when the tumor might be said to be in its incipiency, or when, according to Senn, the tumor is a purely local affection. Removal after infection has taken place is followed by recurrence, which is generally more rapid and extensive than was the primary tumor. The tissue of origin has been found to be the orbital fat, the capsule of Tenon, the episcleral connective tissue, the periosteum of the orbital bones, the lacrimal gland, or the sheath of the optic nerve. Practically it makes little difference from what individual structure the tumor springs, as in all cases complete exenteration of the orbit is demanded.

The microscope alone will determine definitely the variety of sarcoma with which we are dealing; and even this will often show combinations and arrangements quite atypical. The melanotic element of a sarcoma is, of course, readily determined.

Osteosarcomata encroaching upon the orbit are, in the majority of cases, not limited to the orbital bones, but have originated elsewhere in the bones of the skull, and have an extensive distribution.

Osteoma.—Such a tumor is defined by Sutton as an ossifying chondroma. The term should be limited to a true tumor, and not used, as is often the case, to designate those conditions which, though clinically similar, are pathologically not true tumors. Exostoses due to the ossification of existing previously normal structures or to calcification of inflammatory products are histogenetically not tumors. Osteomata are of two kinds, and structurally resemble the shaft of the long bones and cancellous bone-tissue,

respectively. They are thus *compact* or *cancellous*. They are essentially benign, but may cause death or less disastrous results mechanically, attaining eventually great size, though growing very slowly. Pain is a symptom only when they press upon sensory nerves. Compact osteomata are those found in the orbit. They show a preference for the frontal sinus and the roof of the orbit, but may occur in any portion of the orbital walls. If in the roof, the eyeball is pushed downward. They are exceedingly dense, resembling ivory. Their removal from this situation must be undertaken with great care, as in the operation the anterior cerebral fossa may be easily opened, with serious consequences. Their removal is best accomplished by completely denuding the tumor of periosteum, then drilling through its base a number of holes, after which careful manipulation with mallet and chisel will, more or less perfectly, remove the growth.

The diagnostic points of osteomata are their extreme hardness, their manifest attachment to bone, perfect fixity, absence of pain in the tumor itself, and slowness of growth. Osteomata originating in the sphenoidal sinus tend to extend backward into the cranium. Some few cases of osteoma exfoliate spontaneously, leaving very extensive disfigurement.

Plexiform Neuroma.—A plexiform neuroma is a benign tumor composed essentially of an overgrowth of the connective tissue of a nerve-sheath. More especially is the endoneurium involved. Thus the tumor lies along a nerve-trunk or in a plexus of nerves. It is of slow growth and painless. In the orbit it is rare, but few cases having been reported. In those the situation was in the upper, outer angle of the orbit, near the lacrimal gland, displacing the eyeball downward, inward, and forward. If it is an extension from a tumor lying in the skin around the orbit, the diagnosis can be made with much greater certainty. It should be completely removed.

Epithelial Tumors.—These are represented by three great classes, namely: *papillomata, adenomata,* and *carcinomata.* Within the orbit they do not primarily occur, except adenomata and carcinomata in connection with the lacrimal gland and the eyeball, and are therefore to be considered in the chapters dealing with these structures. Secondary invasion of the orbit by epithelial cancer originating in the skin of the lids, or in one of the adjacent cavities, is not infrequently met with. The destruction wrought is often very great, as in a case recently operated on in the German Hospital, in Philadelphia, in which after exenteration the floor, the roof, and the inner wall of the orbit were found to be destroyed.

Cysts of the Orbit.—True cysts consist of an abnormal dilation of pre-existing cavities. Under this restricted definition we find in the orbit

1. Cysts in connection with certain bursæ situated around the superior oblique muscle, where its tendon passes through the trochlea, and also between the levator palpebræ and superior rectus muscles. These cysts have been described as *hygromatous degeneration* of the orbital bursæ—*exudation cysts.*

2. The cystic condition sometimes found in connection with microph-

thalmos and anophthalmos. Such cysts are situated in the lower lid and extend deep into the orbit.

3. Retention cysts, or some of those called by de Wecker *follicular cysts,* are thought by him to have their origin in the follicles of the skin. They may extend into the orbit.

4. Cystic degenerations of the lacrimal gland, which are considered in the chapter treating of that organ.

5. Extravasation cysts, which are often spoken of as hematoceles and hematomata,—blood-cysts,—are exceedingly rare, if not of doubtful occurrence in the sense of having a true cyst-wall and bloody contents. Dermoids are sometimes very vascular, and, according to Berlin, puncture for diagnostic purposes may cause hemorrhage within them. Such a condition might be mistaken for a blood-cyst, but the true character of the affection would remain the same.

There are two cystic conditions found in the orbit of parasitic nature, namely: *hydatid,* due to the *echinococcus,* and the *cysticercus cellulosæ.* These conditions do not fall within the above definition of true cysts. They occur wheresoever the parasites lodge, and make for themselves space by pushing aside tissue, and not by the dilation of pre-existing cavities.

Dermoids are sufficiently distinctive in origin and history to be classed alone. Their presence in the orbit has been mentioned on page 619.

All cysts and cystic conditions within the orbit are, *per se,* innocent. The one general characteristic symptom is fluctuation. Since this is common to all cysts, it simply indicates the presence of contained fluid, and does not aid in a differential diagnosis as to the kind of cyst. It must not be forgotten that cystic degeneration may take place in malignant tumors, and might thus give rise to an error in prognosis. The other symptoms are those common to all growths within the orbit, but often with variations in degree. For instance, immobility of the eyeball in case of malignant orbital tumor often amounts to absolute fixity, whereas cysts generally cause only a restriction of movement.

Exploratory puncture, bringing to examination the contents, is of great value in determining the nature of the cyst. The presence of scolices will show at once that we are dealing with the echinococcus. The aspiration of cerebro-spinal fluid requires a very careful and critical examination of the case before further procedure. The condition is either a meningocele or encephalocele, or an obliterated meningocele: *i.e.,* one in which the neck of the sac has been constricted and obliterated, leaving the sac, with its cerebro-spinal fluid contents, in the orbit, but no longer connected with the brain. In the first instance non-interference is imperative; in the second removal may be attempted with impunity.

Talko has reported a case of congenital cyst situated deep in the orbit of a 3-months-old child, pushing the eyeball forward. Operation was attempted and the anterior portion of the cyst was removed. Serous fluid escaped for ten days, when the child died. The finger could be pushed

into the inferior orbital fissure and into the optic foramen. He believed that it was, beyond a doubt, a posterior meningocele.

The diagnosis of the cysticercus is problematic until after removal. Ciliary neuralgia is said to be rather a constant symptom of echinococcic cyst.

TREATMENT.—Orbital cysts may be treated by excision, incision, injection, or electrolysis. Complete excision, when possible, is preferable. When this is not practical, incison, liberation of the cystic contents, and destruction of the sac, so far as possible, with the curette or by the injection of iodin should be tried. The writer has had no experience with electrolysis.

INFLAMMATIONS OF THE ORBIT.

Periostitis.—Like the same process elsewhere, periostitis of the orbital bones may be acute or chronic, localized or diffused, and is characterized by deep-seated pain and swelling. Secondary to the swelling the neighboring intra-orbital structures may become involved. Thus swelling and edema of the lids and conjunctiva, limitations in the movements of the eyeball, and proptosis and other malpositions of the globe are produced. The patient may complain of diplopia. Pressure on the optic nerve may give rise to neuritis, demonstrable upon ophthalmoscopic examination, or atrophy of the nerve may be caused by direct pressure.

ETIOLOGY.—Traumatism, rheumatism, syphilis, and scrofula are the potent etiologic factors, syphilis, either acquired or congenital, being the most frequent.

SYMPTOMS AND COURSE.—Acute periostitis, if extensive, may be ushered in by a chill, followed by a rise of temperature. The patient will experience pain and distress, often severe in character, located in and around the orbit; and will frequently complain of frontal headache. If the inflammation is deeply seated, movements of the eyeball and pressure backward will cause pain within the orbit. If the orbital margin is affected, the overlying skin will be swollen, red, and adherent, the pain and tenderness on pressure being at times exquisite. The pain is generally worse at night.

The course of the disease is that of any acute inflammation. It may terminate in resolution with complete and rapid recovery; or abscess formation may supervene with a variable collection of pus, undermining and detaching the periosteum and thus encroaching more upon the orbital space; or it may result in bone destruction (caries and necrosis), with the formation of sinuses and sequestra, which carry the disease into chronicity. Chronic periostitis may last for months or years, while intractable sinuses, retraction of the skin with alteration in shape and mobility of the eyelids, set up a train of symptoms which may continue long after the original disease has subsided.

Caries generally attacks the margin of the orbit, and is most frequent in syphilitic and scrofulous children, whereas necrosis is most frequent in

the adult and follows acute periostitis or traumatism, such as fracture denuding the bone of its periosteum. Large portions of bone may become exfoliated and great deformity may result.

The location of orbital periostitis, whether marginal or deeply seated, is of great importance to the patient, the most dangerous points of attack being at the apex and at the roof. At the apex is the optic nerve, with its rather vulnerable sheath; pressure here may cause neuritis or rapid atrophy of the nerve. The extra-ocular muscles, with the exception of the inferior oblique, have their origins at this point, forming the apex of the muscle-cone, while beneath the external rectus muscle is the ciliary ganglion. Therefore the pressure, if severe or long continued, may cause permanent paralysis of one or more of these muscles. The roof of the orbit is thin and easily destroyed, in which event direct communication would be established with the anterior cerebral fossa. The danger of meningitis would then be imminent. Fortunately the most frequent situation of orbital periostitis is near the margin, up and out.

Diagnosis.—This is generally not difficult; the boring, deep-seated pain, with nocturnal exacerbations, together with the external signs, serve to implicate the periosteum in the inflammatory process. To determine, however, to what extent other structures are involved; or, if there be, for example, tenonitis, cellulitis, or orbital phlegmon, which structure is primarily affected may be difficult or uncertain. Tapping with the finger around the orbital margin, so as not to disturb the orbital contents, will often serve to locate the affection in the bones. If this can be done without pain, the bone or periosteum is probably not involved. *Vice versa*, if gently pushing back the eyeball, with just sufficient force to reach the soft parts only, causes marked pain, we probably have to deal with tenonitis or cellulitis.

Syphilitic periostitis is generally a tertiary manifestation, but it should be remembered that it may also be found in the complex of secondaries. Noyes has noted the fact that immobility of the eyeball due to periostitis is to be differentiated from ophthalmoplegia of third-nerve origin by the absence of ptosis. The third nerve divides at its entrance into the orbit into an upper and a lower portion. The upper division supplies the levator muscle, and has a well-protected course between that muscle and the superior rectus, entering the lower surface of the muscle.

If sinuses be present, evidencing necrosis or caries, great care and gentleness must be used in probing lest the bone be further injured. Especially easy is it to perforate the necrotic bone at the roof of the orbit and pass the probe into the cranial cavity, often with serious results.

Prognosis.—The prognosis of acute orbital periostitis is favorable, providing the process can be checked before irreparable damage has been done to the intra-orbital contents. Chronic periostitis may last for months and years with the formation of intractable sinuses and occasional exfoliation of bone, or with established communication with the adjacent cavities.

Gummata occasionally attack the periosteum of the orbital bones, though they occur much less frequently here than in the other cranial bones. The condition is a rarefying osteitis: *i.e.*, the subperiosteal bone-cells undergo softening and become in structure similar to the gummatous tissue. The symptoms are those of ordinary periostitis with certain exaggerations. Nocturnal pain and neuralgia are prominent symptoms. The swelling is more circumscribed than in periostitis and simulates more a true tumor, exophthalmos being frequently produced if the gumma is in the deeper parts of the orbit. Orbital gummata cause great immobility of the eyeball, marked fixity of the eyeball being characteristic of this form of inflammation. They are amenable to specific treatment, but when they disappear great holes and depressions are left in the bones.

TREATMENT.—Orbital periostitis requires constitutional and local remedies. Constitutionally the treatment depends upon the cause, if known, and in any event the general nutrition of the patient is of importance. The first stages of the attack, if marked, must be met with depletion and antiphlogistics. A purge, preferably salts, should be given, followed by a fever mixture, if fever be present. If the presence of syphilis is confirmed, mercury and potassium iodid are indicated. If of rheumatic origin, salicylate of sodium and the iodid are indicated. If the patient is scrofulous, iron, codliver-oil, hypophosphites, and appropriate diet are to be considered. For the control of pain salicylate of sodium given in full doses will seldom fail. It is especially useful in aborting or annulling the nocturnal paroxyms.

Locally, the indications are to limit the exudation and promote absorption and resolution. Although the application of cold is generally of use in the first stages of inflammation, practically it is not to be recommended in periostitis, heat being much more efficient as well as more grateful to the patient. Hot fomentations therefore play an important rôle. They must, however, be properly applied, the object being to maintain a uniformly high degree of temperature. Hot compresses replaced every half-minute for thirty minutes, repeated in an hour or two, will generally yield good results. A lukewarm or cold compress placed over the part for an indefinite time is useless.

If pus is present it must be liberated. This is to be done by passing a long bistoury in such a way as to avoid the eyeball, the optic nerve, and the ciliary ganglion. The incision should be made through the periosteum to the bone, and the mouth of the opening should be kept patulous with gauze packing. Sinuses should be carefully syringed, and any loose piece of bone presenting should be removed. It is well to form a definite idea of the size and extent of the loose bone before attempting its removal, always bearing in mind the proximity of the cranial cavity, the frontal and ethmoidal sinuses, and the antrum.

Periostosis is a thickening of the periosteum following inflammation. It is said to occur in those cases in which the bone proper has not been

sufficiently diseased to induce caries. It more frequently affects the margin, and is generally syphilitic in origin. It may attack the periosteum at the apex, in which case permanent, but slowly produced, paralyses of the ocular muscles, or even atrophy of the optic nerve, may result. At the orbital margin neuralgia from pressure of the supra-orbital or infra-orbital nerves is a frequent symptom. The treatment is antisyphilitic and absorbent, mercury and potassium iodid yielding the best results.

Hyperostosis of the orbital bones is an exceedingly rare condition, having been noted by Bull but four times in about twenty thousand cases. It is characterized by an increase in the diameter of the bone, and is primarily a disease of the bone itself. Such a condition could, of course, cause marked and curious deformity. Syphilis is not thought to play much of a part in the causation.

Exostosis is usually due to syphilis, and may occur in any portion of the orbital wall. It is often caused by a long-continued chronic periostitis. Exostoses may grow into the orbital cavity and produce pressure symptoms. They grow more frequently, perhaps, from the os planum of the ethmoid than from other areas. They are of more abrupt elevation than the swellings of periostosis, and partake more of the nature of a tumor with better-defined limitations. They will sometimes yield to antisyphilitic treatment. If they do not yield to such treatment, but become destructive in their progress, they must be surgically removed. They should be attacked at the base with chisel and mallet. The chiseling must be done very carefully so as to not fracture the bone. It is best to use the corner of the edge of the instrument, progressing slowly, with repeated short strokes.

Orbital Cellulitis and Phlegmon.—Orbital cellulitis, or inflammation of the fatty cellular tissues within the orbit, may occur as an acute, as a subacute, or as a chronic inflammation. All grades of severity are recognized, from the mildest, with only local symptoms and a course tending toward rapid resolution and recovery, to the phlegmonous variety with grave constitutional disturbance, thrombosis of the cavernous sinus, and extension to the brain, the latter causing purulent meningitis and death.

It may follow traumatism, such as a blow, punctured wounds, or fracture of the orbital bones, or it may be an extension from local pyogenic processes in neighboring parts. It has been seen after laceration of the scalp caused by unskillful use of the obstetric forceps. Long exposure to cold is sometimes a cause.

Among other causes of this condition are periostitis; meningitis complicated by thrombosis of the cavernous sinus and ophthalmic vein; panophthalmitis, in which the process passes beyond Tenon's capsule; suppuration in the ethmoidal cells or around the upper teeth and upper jaw; and very rarely dacryoadenitis. Typhoid and typhus fevers, scarlatina, pyemia, and puerperal septicemia, and especially facial erysipelas may all cause orbital cellulitis. When it occurs in the course of these diseases it must be regarded as a very serious and often dangerous complication.

Symptoms.—The symptoms, in mild cases, are dull pain and headache; restricted ocular movements, with perhaps slight proptosis and diplopia; swelling of the lids, and edema of the conjunctiva (chemosis). In the phlegmonous form there is an exaggeration of these symptoms together with constitutional disturbance and other symptoms due to pressure.

The attacks may commence with a chill followed by a fever. The lids are greatly swollen, tense, and dusky, all the folds and landmarks being obliterated. The eyeball is pushed far forward, and is absolutely fixed. The chemosis is often so extensive as to hide completely the cornea, which

Fig. 363.—Abscess of the orbit in a child ten days old, injured during instrumental delivery. (Author.)

may become devitalized and slough. Pressure on the optic nerve will cause neuritis and atrophy. Such a condition of congestion and stagnation is generally attended by more or less thrombosis of the veins which may continue into the cavernous sinus, and in some cases may extend to the sinus of the other side, and thus affect secondarily the fellow-eye. If allowed to run its course, pointing finally takes place, followed by rupture and the liberation of pus. The point of rupture is usually through the upper lid near the orbital margin.

Prognosis.—The milder cases are amenable to treatment, and the prognosis is good, complete recovery being the usual termination. In graver

cases the prognosis as regards the life of the patient is favorable. Should meningitis occur, a fatal termination is to be expected. As regards the future usefulness of the eye, prognosis is to be based upon the amount and duration of pressure and the extent to which the eyeball has taken part in the inflammation. Sloughing of the cornea, optic-nerve atrophy, thrombosis in the retinal veins, and a development of pus within the eyeball are all fatal to vision.

TREATMENT.—This should be along the lines of general surgery. The patient should be sent to bed. To limit exudation cold is indicated in the earliest stages. To aid absorption and resolution heat should be applied thereafter. Hot stupes give great relief and are much to be depended upon. Depletion by the application of leeches to the temple is often of service in the commencement of the attack. Pus must be liberated at once, for it can work its way backward, with disastrous results. As it is generally present long before it manifests itself by pointing, when the symptoms are severe, its presence should be presumed, and free incisions should be made deep into the orbit under strict antiseptic precautions. Even though no pus be found, the local bloodletting will fulfill an important indication. The incision should be made with a long, straight bistoury—by preference, through the conjunctiva. If, on account of swelling and edema, this should be impracticable, the incision can be made through the upper lid. The knife should pass close to and parallel with the orbital wall, avoiding the eyeball and the optic nerve. The opening of the incision should be freely enlarged, a bichlorid-of-mercury solution (1 to 3000) should be carefully introduced by syringing, and drainage should be maintained by loose packing. The general treatment is tonic and supportive. Good diet, iron, quinin, strychnin, and alcohol in the form of milk punches are indicated.

Inflammation of the Oculo-orbital Fascia (Tenonitis).—Inflammation of Tenon's capsule is sometimes found as a distinct affection and always is a part of severe orbital cellulitis, in which case, however, it is scarcely to be differentiated from the more general inflammatory process.

SYMPTOMS.—The symptoms include slight or moderate swelling and edema of the upper lid, which, on account of the firm attachment of processes of the orbital fascia to the upper margin of the tarsus, may be more marked in the lid above the tarsus. Chemosis, pain on motion, and restricted movement of the eyeball, with some proptosis, are present. These symptoms are all found in orbital cellulitis, in which condition they are generally more pronounced. The most characteristic symptom of tenonitis, and the one which, when present, establishes the diagnosis, is the occurrence of vesicular swelling over the insertion of one of the straight muscles. It will be remembered that the straight muscles pierce Tenon's capsule by a kind of invagination, thus offering a favorable situation for the pushing forward of serous fluid collecting within Tenon's space.

ETIOLOGY.—Tenonitis may follow operations directly disturbing the capsule, such as tenotomies and advancements. It is then usually severe and,

if suppurative, endangers the integrity of the eyeball. Bull mentions a case known to him of double panophthalmitis resulting in blindness from a simultaneous tenotomy of both internal recti muscles. Such a calamity sounds a note of warning against the slightest carelessness in the performance of these operations, however simple and usually free from danger they seem to be. Zentmayer reports tenonitis as a complication in a case of typhoid fever. It has been known to occur after diphtheria and influenza. The most frequent causes of idiopathic tenonitis are rheumatism and gout. These diseases are often responsible for episcleritis, with which certain cases of tenonitis might be confounded. Ordinary care in the examination should make the differential diagnosis clear.

PROGNOSIS of the traumatic cases must be guarded, the danger of panophthalmitis being imminent in any pyogenic inflammation of the capsule. The other cases, as a rule, recover, leaving the parts functionally normal. The duration depends upon the character of the inflammation, and in chronic cases may be many months.

TREATMENT.—This does not differ from that of the preceding affection. If pus is present, free incision into the capsule for its exit must at once be made. This is an important procedure, and must be unhesitatingly accomplished. Salicylic acid, or salicylate of sodium, potassium iodid, or colchicum is indicated if the cause is rheumatism or gout.

Diseases of the Frontal, Ethmoidal, Maxillary, and Sphenoidal Sinuses.—These cavities or air-spaces are lined with mucous membrane, and are liable to inflammation by extension from the nasal mucous membrane or to retention disturbances due to occlusion of their natural outlets. The two frontal sinuses are usually completely separated from each other by a thin bony partition. They are not developed until about the sixth or seventh year, and vary greatly in size in different individuals. Tilley, after an examination of one hundred and twenty skulls, concludes that the normal frontal sinus should measure twenty-eight millimetres from the median line outward, and twenty to twenty-eight millimetres in its vertical dimension. Some sinuses he found only large enough to contain an ordinary bean, while others had a capacity ten times as great. The frontal sinuses communicate with the anterior ethmoidal cells, and in common with them with the nasal cavity at the middle meatus, through the infundibulum. The anterior and posterior ethmoidal cells do not communicate with each other, being separated by a bony partition. The posterior ethmoidal cells are sometimes in communication with the sphenoidal sinuses. They open into the superior meatus of the nose.

The maxillary sinus, situated in the body of the superior maxilla, communicates with the middle meatus of the nose. Its roof is the thin orbital plate of the superior maxilla, forming the floor of the orbit. In some cases the roots of the first and second molar teeth perforate the floor of the antrum. The floor of the antrum may be in relation with all the teeth of the true maxilla from the canine to the wisdom-tooth (Sattler).

The sphenoidal sinuses do not exist in early childhood. When developed they are very irregular in size and in shape. They communicate with the superior meatus of the nose, and, as mentioned above, occasionally with the posterior ethmoidal cells.

Inflammation in these sinuses generally starts from the nose, a catarrhal rhinitis, for example, extending through the infundibulum to the frontal sinuses and to the anterior ethmoidal cells, or from the superior meatus into the posterior ethmoidal cells and sphenoidal sinuses, or from the middle meatus into the antrum. The antrum may also become infected through the teeth, which in many cases perforate its floor.

The first stage is a thickening and congestion of the mucous membrane, a narrowing of the natural outlet, and a retention of mucous secretion within the sinus. This condition is known as *mucocele of the sinus.* Following this there may be great distension of the cavity; the orbital space is encroached upon, and ocular symptoms make their appearance. The final stage is that of suppuration. The process remains no longer limited to the sinus; orbital abscess may ensue, with all its dangers, or extension backward or upward into the brain-cavity may cause a fatal meningitis.

Without prompt and skillful treatment necrosis of the bones and the formation of intractable fistulæ may generally be expected in these cases.

Tumors and polypi may spring from the mucous membrane of the accessory sinuses and push their way into the orbit, or the sinuses may become secondarily invaded by tumors originating elsewhere.

Symptoms and Diagnosis.—There are certain symptoms of the diseases of the accessory sinuses in their relation to the orbit which are common to all the cavities. The first stage of the disease is marked by pain on pressure or percussion over the affected sinus. If this is the frontal sinus, headache above the root of the nose is an almost constant symptom. There may be neuralgic pain over the course of the supra-orbital nerve and a discharge of mucus from the corresponding nostril.

In ethmoiditis headache is less constant, and mucous discharge from the nostril perhaps is more constant. The pain in antral disease is located in the upper jaw, and sometimes over the course of the infra-orbital nerve.

It is impossible to diagnose disease of the sphenoidal sinuses in the first stages. If pus has formed, its presence in the superior meatus of the nose is of diagnostic value.

In the second stage, or that of distension, the diagnosis is easier. Distension of the frontal sinus generally manifests itself at the upper, inner angle of the orbit. There is a rounded swelling between the trochlearis pulley and the angle of the orbit, pushing the eyeball downward and outward. Distension of the ethmoidal sinuses causes exophthalmos and a displacement of the eyeball outward. If the antrum become distended, the swelling generally presents in the floor of the orbit directly back of the orbital margin, giving rise to exophthalmos, with upward displacement of the eyeball. From the situation of the sphenoidal sinuses, as might be sup-

posed, their distension may cause sudden blindness from pressure on the optic nerve. The exophthalmos would be directly forward.

Of late the means for transillumination have been greatly improved, making it possible in many cases to determine with facility the presence of collections within the sinuses. They may all be transilluminated except the sphenoidal.

The third stage of empyema, unless free drainage is obtained, is likely to be complicated by orbital abscess, the symptoms of which have been described. As the bones become thin from distension, fluctuation may often be elicited, thus distinguishing the condition from solid tumor.

Among the reflex symptoms which may exist in disease of any of the sinuses are lacrimation, photophobia, reduced vision, restricted fields for form and color, and relative scotomata. Diplopia is present in accordance with the displacement of the eyeball. Muscle paralysis and optic neuritis will correspond to the amount and position of the pressure.

ETIOLOGY.—The majority of cases have their origin in the nasal cavity. Such an inflammation may occur in erysipelas, epidemic influenza, typhoid and other infectious fevers, tuberculosis, or syphilis. Fracture of the bones may so alter the anatomy as to cause obstruction of the infundibulum, damming back the mucous secretion in the frontal sinuses and the ethmoidal cells. Disease of the teeth in relation with the maxillary sinus may be the exciting cause of antral disease.

TREATMENT.—In the early stages this belongs to the rhinologist. In many cases the swollen mucous membrane at the openings of these sinuses into the nose may be effectually treated with adrenal solution, or other drugs with similar action, and the normal outlets thus can be kept open. In the later stages, on account of the ocular symptoms, these patients are generally brought to the ophthalmologist. The indications are, first, to establish a free communication with the cavity, and, second, through this communication to maintain drainage and cleanliness. In the case of the frontal sinus an opening is to be made with a stout knife through the thin and distended bone at the upper, inner angle of the orbit. Then, with the finger in the sinus and a gouge up the nose, a second opening may be forced. A fenestrated drainage tube is then to be passed from the sinus into the nose, through which careful and repeated syringing of antiseptic solutions may be carried on.

In ethmoiditis the incision is to be made through the os planum of the ethmoid, and a passage for a drainage tube is to be forced into the nose.

The antrum may be reached through the canine fossa of the malar bone or through the socket of the second molar tooth. If the floor of the orbit is thin and bulging, an opening may be made here, taking care not to injure the infra-orbital nerve. Much time is required for the treatment of these cases. It will often be necessary for the patient to wear a drainage tube and to submit to syringing of the cavity several times daily for weeks or months.

Cavernous-Sinus Thrombosis.—Thrombosis of the cavernous sinus as a primary affection rarely occurs. When present, it is usually the result of extension of a thrombotic process in the orbital veins, or a similar condition in the other sinuses.

SYMPTOMS.—Whether marasmic or inflammatory (septic), the local symptoms are the same and are, first, those of venous stasis; second, those of pressure; third, those of paralysis.

Individually considered they are edema of the lids, which may extend for some distance into the integument of the face; chemosis; and exophthalmos, which, at first slight, generally becomes pronounced. The eyeball is more or less mechanically restricted in its movements, and if the upper lid does not from edema obstruct vision on that side, diplopia is complained of. Interference with the cranial nerves running to the orbit is likely to occur. The third, the fourth, the ophthalmic division of the fifth, and the sixth nerves all traverse the cavernous sinus, and are liable to injury. The one most commonly affected is the third. We would then have divergent strabismus, stabile mydriasis, and ptosis. Paralytic ptosis, although a rather constant symptom, is often difficult to establish, on account of the mechanical ptosis due to edema of the lid. Other varieties of strabismus would, of course, occur were one or more of the other nerves paralyzed. At the commencement of the attack irritation preceding the paralysis would cause miosis. This, however, is of short duration. As in all conditions of retrobulbar pressure, the optic nerve may suffer neuritis or atrophy. Through the medium of the circular sinus the process may extend to the opposite side. According to Macewen, this occurs in more than half the cases. Such a transference is pathognomonic, and serves to diagnose positively cavernous-sinus disease from intra-orbital disease with many symptoms in common. Another important diagnostic sign of cavernous-sinus thrombosis, when present, is edema over the mastoid region. Its occurrence has been explained as being due to stasis in the emissary vein of Santorini, which empties into the lateral sinus, which, in turn, directly communicates with the cavernous sinus.

Owing to the free anastomosis of the orbital veins with the veins of the face, blood from the orbit has an extra- as well as an intra- cranial means of exit. To this arrangement is due the fact that certain cases of cavernous-sinus thrombosis will present orbital symptoms not commensurate with the amount of obstruction in the sinus. Such cases are of more gradual development, and favor the presumption that in them the process is extending from within outward, rather than from without inward, thus giving more time for the formation of an adequate collateral circulation.

Thrombosis of the ophthalmic vein or of the cavernous sinus is accompanied by pain, notably over the course of the supra-orbital vein and in the eyeball. The patient complains of varying degrees of headache, and, if the case is complicated with meningitis or brain-softening, cerebral symptoms are apparent.

ETIOLOGY.—Two forms of thrombosis are recognized—marasmic and inflammatory. The first is due to an impoverished condition of the blood and occurs usually at the extremes of life or as a complication of the exanthemata and other diseases producing marked debility. Inflammatory or septic thrombosis is caused by an alteration in the vessel-wall (phlebitis, meningitis), metastatic transference of inflammatory or septic products, or traumatism. Orbital cellulitis is almost invariably accompanied by more or less thrombosis, which may readily extend into the cavernous sinus. Caries and necrosis of the bones of the skull or around the teeth have been responsible for some cases. Fracture of the skull—especially at the base, chronic otitis media, erysipelas, furunculosis, or pyogenic foci so situated as mechanically to infect the blood *en route* through the orbit and cavernous sinus are all recognized causes.

Fig. 364.—Thrombosis of the cavernous sinus. (W. T. SHOEMAKER.)

PROGNOSIS.—The disease may last from a few days to several months. According to Macewen, the great majority of cases of cavernous-sinus thrombosis end fatally. This author further says that absolutely all cases of infective thrombosis end in death.

TREATMENT.—This must follow general surgical principles, meeting exigencies as they arise. Hartley opened by trephine the cavernous sinus in a case diagnosed by Knapp, and removed the thrombus. This operation has been performed several times since.

Hemorrhage into the Orbit.—In the vast majority of cases hemorrhage into the orbital cellular tissue is caused by traumatism, either direct or indirect. Instruments or foreign bodies thrust into the orbit must necessarily cause bleeding. Blows upon the eyeballs or head or fracture of the skull involving the orbital bones may permit of an outpouring of blood

into the cellular tissue. The condition rarely occurs spontaneously, and may then be due to hemophilia, scurvy, deterioration of the blood-vessels, or to too great a rise of blood-pressure incident to coughing, sneezing, straining, etc. Once liberated within the orbit, the course of the blood is determined by the layers and processes of the orbital fascia. It extravasates in the direction of least resistance, which is generally forward, and in a short time comes to view under the conjunctiva of the eyeball or eyelids. According to the amount of hemorrhage the symptoms will vary from those scarcely noticeable to marked proptosis, limited ocular movements, great subconjunctival accumulation of blood, and pressure injury to muscles and nerves, including the optic nerve, with perhaps loss of vision.

The diagnostic value of subconjunctival hemorrhage after injury, in determining fracture at the base of the skull implicating the orbital bones, has depreciated somewhat since the demonstration a number of times by autopsy of traumatic orbital hemorrhage without fracture. Evidence of orbital hemorrhage is, notwithstanding, a valuable and serious symptom after head-injury, and makes basal fracture at least probable. Sometimes the blood escapes into the nose, indicating fracture of the ethmoid bone.

A few cases of spontaneous subperiosteal orbital hemorrhage in infants suffering from malnutrition have been reported by Spicer, and a so-called traumatic subperiosteal blood-cyst has been observed by Baquis, which disappeared within four days. Denig reports a case of subperiosteal blood-cyst situated behind the lacrimal gland, extending toward the orbital roof, where it had caused perforation into the anterior cerebral fossa. The patient had received a severe orbital injury ten years before.

PROGNOSIS.—Insomuch as the optic nerve is always jeopardized by orbital hemorrhage if extensive enough to cause considerable pressure, the prognosis should be guarded *quoad visus* in the presence of pressure symptoms.

TREATMENT.—Absorption takes place in from three to six weeks and may be aided by hot fomentations. A pressure bandage may be indicated in certain cases, and, when the proptosis is so extensive as to prevent closure of the lids, the cornea must be carefully guarded. The use of the bistoury for the purpose of liberating the blood may be advisable when the bleeding has been excessive, but in ordinary cases it seems to the writer to be of doubtful efficacy, as extravasations are not often relieved by incisions.

Pulsating Exophthalmos.—In typical cases this condition is characterized by protrusion of the eyeball; noises or bruit to be heard at the entrance of the orbit, or by auscultation over various points of the skull, even over the occiput; and by pulsation near the orbital apex transmitted through the eyeball. The exophthalmos, if unilateral, or if more pronounced on one side than on the other, gives rise to diplopia. The bruit is both a subjective and an objective symptom. It is increased by lying down or stooping, or by any exertion raising the blood-pressure. The noises in the head are incessant and cause much distress. They stop or are greatly

modified when the blood-supply to the side of the head affected is interrupted by compression of the common carotid artery. The pulsation—generally demonstrable by careful palpation—is sometimes visible. The veins of the lids, of the conjunctiva, and of the retina are often distended and tortuous—the result of passive hyperemia. In a certain number of cases optic neuritis, optic-nerve atrophy, retinal hemorrhages, glaucoma, and cataract have been noted. More frequent than these last-named symptoms is paralysis of the abducens nerve.

ETIOLOGY.—Exophthalmos with pulsation may be due to a variety of lesions, both intra-orbital and extra-orbital. Thus, it may be a symptom of extremely vascular tumors within the orbit, due to a true aneurism of the ophthalmic artery in its intra-orbital or intracranial portion, aneurism of the internal carotid artery, arterio-venous communication, varicose dilation of the orbital veins, or communication between the internal carotid artery and the cavernous sinus. The last-named condition has been found to be the most frequent cause. Thus, the disease in the majority of cases is of intracranial origin, the protrusion of the eyeball and the other orbital symptoms being secondary and dependent upon venous obstruction. Rivington demonstrated the intracranial origin of pulsating exophthalmos, which was a distinct advance in the pathology of this condition. In an analysis of 19 autopsies Frost found orbital aneurism in 3, affection of the cavernous sinus in 2, aneurism of the intra-orbital portion of the ophthalmic artery in 2, arterio-venous communication in 8, and the condition undetermined in 4.

With arterio-venous or arterio-sinus communication we have obstruction of the venous outflow of blood, which induces marked distension and varicosity of the veins. The blood-current then becomes reversed, and the distended veins carry arterial blood (Sattler). At this stage pulsation commences. As more or less time is required for these changes to take place, and as, indeed, they may not take place, pulsation may not appear early nor need it occur at all. Bilateral pulsating exophthalmos of traumatic origin is to be explained by a transference of the obstructing process from the cavernous sinus of one side by way of the transverse and circular sinuses to the cavernous sinus of the opposite side.

The majority of cases are traumatic; according to de Schweinitz, 60 per cent.—110 in 181 cases. Punctured wounds, gunshot injuries, and fracture at the base of the skull are the chief traumatisms. Rupture of the internal carotid artery into the cavernous sinus is generally due to basal fracture. Other cases are idiopathic.

DIAGNOSIS.—The diagnosis in typical cases is not difficult, but the exact character of the lesion is often impossible to determine. Vascular tumors offer more resistance to pressure than do aneurismal conditions, and the proptosis caused is less likely to be directly forward. History of a fall or blow on the head with unconsciousness places the lesion probably with the majority: *i.e.*, intracranial. Following penetrating wounds into

the orbit, intra-orbital arterio-venous communication has likely been established. Whether the ophthalmic artery is the seat of an aneurism in its intracranial or in its intra-orbital portion is impossible definitely to determine.

PROGNOSIS.—This is favorable, providing surgical interference is permitted. The distressing subjective symptoms can generally be relieved or completely cured. If the condition is allowed to continue and increase in severity, the eyeball is apt to suffer destruction from secondary glaucoma, or keratitis occurs from pressure on the ophthalmic division of the fifth nerve in its course through the sphenoidal fissure.

TREATMENT.—The most rational and the most radical treatment, and that which promises the best results, is ligation of the common carotid artery. The operation is, however, not unattended by danger to life, although the danger is being minimized by modern surgical methods. De Schweinitz has tabulated one hundred and two cases in which the common carotid artery was tied, with a death-rate of 10.7 per cent. This mortality-rate in comparison with that given by some modern surgeons is extremely low. Digital compression has cured some cases, and probably should be tried first. With compression, full doses of potassium iodid and a restricted diet have been recommended. Spontaneous cure rarely occurs.

De Schweinitz advises the order of procedure as follows: Compression with or without the administration of potassium iodid; in event of failure, ligation of the common carotid. He strongly recommends the early consideration of ligation in the treatment of these cases, believing that compression or medicinal treatment should be regarded as inefficient unless signs of amelioration promptly appear.

Intermittent Exophthalmos is a rare condition in which the eyeball is protruded when the subject stoops or leans forward, and recedes within the orbit when he is erect or in the recumbent position. Or the exophthalmos may be voluntarily produced by the act of blowing. Posey reports a case in which a young adult could in this manner voluntarily proptose the left eyeball fifteen millimetres in advance of its fellow. The cause is presumably some varicose condition of the veins of the orbit.

Enophthalmos.—This condition is a recession of the eyeball within the orbit, and may be idiopathic or traumatic. Idiopathically it is found after absorption of the orbital fatty cellular tissue in the aged, or after long-continued wasting disease, or after rapid extraction of the fluids of the body, such as occurs in Asiatic cholera (von Graefe). These cases are always bilateral. To a slight extent it may often be noted during an attack of migraine; it is then unilateral. Very rarely the eyeball is retracted by the combined and simultaneous action of the four recti muscles—*spastic enophthalmos*. W. Förster has recently reported a case of enophthalmos incident to forcible separation of the eyelids, the eyeball returning to its normal position when no force was applied. The patient was aged and emaciated. Two similar cases have since been reported.

ETIOLOGY AND PATHOLOGY.—The pathology of traumatic enophthalmos has been the subject of much discussion, and as yet is based on a number of theories. Beer believes the lesion to be one of the nerve-centres or tracts, particularly of the sympathetic or trigeminal nerves, causing absorption and atrophy of the orbital cellular tissue. Gessner presumes it to be due to periostitis of some portion of the orbital wall and inflammation of the retrobulbar tissue resulting in cicatricial contraction, the eyeball falling backward owing to the reduced bulk of the orbital contents. Schapringer's theory is that Müller's orbital muscle is paralyzed from injury to the sympathetic nerve, thus increasing the orbital space and permitting of recession of the eyeball. Lang, Tweedy, Langenbeck, and others regard fracture of the orbital wall as the determining cause of traumatic enophthalmos. In this connection a distinction should be made between displacement of the eyeball in certain cases of undoubted and extensive fracture and cases of enophthalmos in which no such fracture is demonstrable. Lederer concludes that traumatic enophthalmos is to be explained by fracture of the orbital wall and hemorrhage. The writer is of the opinion that the orbital fascia and Tenon's capsule proper are necessary to the maintenance of the eyeball in its natural position. Enophthalmos will result from disease or injury of this fascia, if so situated and so extensive as to throw the balance of power in favor of the enophthalmic forces. This may be the case in paralysis of the smooth muscular fibres in the check ligaments, or in nutritional disturbance leading to atrophy or relaxation of the same. It may result from traumatic rupture of the check ligaments or of the orbital attachment of the fascia; or especially liable would it be to follow rupture of the posterior investing sheath of the eyeball. (W. T. Shoemaker.)

SYMPTOMS.—The symptoms of idiopathic bilateral enophthalmos are simply a recession of the eyeballs directly backward, with the characteristic sunken appearance of the eyes. The palpebral fissures are not narrowed.

In traumatic enophthalmos the appearance is different. The eyeball is not only retracted,—it may be directly backward,—but is very often lowered, with the axis directed upward. The palpebral fissure is always narrowed, and the appearance is that of an artificial eye (Nieden). As the visual axes are no longer in accord, diplopia is often complained of, and the excursions of the eyeball may be more or less limited, owing to the altered muscular relations, the radii of muscular action being shortened. Traumatic enophthalmos is permanent.

Exophthalmic Goitre (Parry's Disease; Graves's Disease; Basedow's Disease).—The three cardinal and characteristic symptoms of this disease are tachycardia, enlargement of the thyroid gland, and exophthalmos. The secondary symptoms are tremor, excessive sweating, nervousness, mental depression, apprehension, emaciation, pain and weakness in the extremities, brittleness of the nails, loss of hair (including eyelashes and eyebrows), increased lacrimation, conjunctivitis, diminished power of convergence, and other less common symptoms. Tremor is a very constant symptom, al-

though at times coming on only late in the disease, and has been classed by some writers as a cardinal symptom (Butler).

Of the three cardinal symptoms, tachycardia is almost constant, and is the first to appear. One or both of the other symptoms may be absent for a long time. According to Gowers, exophthalmos is absent in about one-tenth of the cases and the goitre in about one-twelfth. The heart's action always reaches 100 per minute, and has been frequently noted at 200.

The goitre may be unilateral or bilateral; but if bilateral it is generally more marked on one side than on the other, and commences on that side. The exophthalmos may likewise be unilateral or bilateral, but is generally bilateral. It usually corresponds in side to that of the goitre, but several cases have been reported in which the goitre was on one side and the exophthalmos was on the opposite side. All degrees of exophthalmos are found, and very rarely actual dislocation of the eyeball has occurred. Many patients complain of an intolerable sensation of heat, which may lead them to cast aside their clothing and enables them to withstand low degrees of external temperature. The disease is one of early adult life, and principally affects females. Higgins places the percentage of cases occurring in women at 95 or 97 per cent. This is probably too high. Butler states the ratio to be 3 to 1. The same author calls attention to a family tendency.

The special eye symptoms of exophthalmic goitre are important and interesting. Three phenomena affecting the lids are classic, and should be carefully sought for, although they are not always to be found.

Von Graefe's Sign consists of a want of a proper co-ordination between the upper lid and the eyeball, when the latter is rotated downward. Normally, in this action of the eyeball the upper lid follows proportionately, the amount of cornea covered by the lid remaining the same. In Graves's disease the lid does not so follow; the cornea passes from under the lid, and a line of sclerotic becomes visible above. Lewin has observed this phenomenon in 55.5 per cent. of the cases of Graves's disease, Hill Griffith in 13.2, Pässler in 17.6, and West in 14 per cent. of the cases. Fitzgerald has noted 4 cases of Graves's disease with one-sided exophthalmos, and von Graefe's sign corresponding to that side. Hack has seen 1 case of unilateral exophthalmos with von Graefe's sign, in which the latter disappeared with the former.

Wilbrand and Saenger have collected 39 cases of Graves's disease classified as follows: Exophthalmos in 37 cases—double in 33 and one-sided in 4. Of the cases of bilateral exophthalmos, von Graefe's sign and Stellwag's sign were found in 5. Von Graefe's sign was positive and Stellwag's sign was questionable in 14, and both "signs" were questionable in 4. In all 4 cases of unilateral exophthalmos both "signs" were found affecting the proptosed eye. In 6 cases exophthalmos and both "signs" were not to be found. Von Graefe's sign and Stellwag's sign without exophthalmos were found in 1 case, and in 2 cases Stellwag's sign alone was found. Sharkey, in 613 cases, found von Graefe's sign in 601.

Several theories have been advanced for the explanation of this lid phenomenon. Von Graefe believed it to be due to stimulation of the sympathetic nerves whereby the fibres of Müller's palpebral muscle were contracted, thus holding the lid back. Dilation of the pupils, which should follow stimulation of the cervical sympathetic, is not, however, an accompanying symptom.

Sattler's explanation is a disturbance of the reflex and co-ordination centres (association centres). Against this theory has been urged an insufficient knowledge of these centres to warrant its acceptance. Also that long-continued disturbance of these centres should produce lasting results, which is not the case.

Contraction of the levator palpebræ muscle brought on by increased blood-supply is the theory of Ferri; and an insufficiency of the orbicularis is that of Sharkey.

Möbius and Bruns hold that in Graves's disease there is an increased activity or power of the eye-musculature—an hypertonus of the levator muscle. Möbius believes that the primary phenomenon is Stellwag's sign, and that this is followed by von Graefe's symptom.

Last must be mentioned the mechanical theory of Wilbrand and Saenger, which seeks to explain the phenomenon by a retrograde or reversed action of those muscle-fibres connecting the levator palpebræ and the superior rectus muscles, which also have connection with the fornix of the conjunctiva. Normally, when the eyeball is rotated downward, traction made upon the fornix is transmitted through the above-mentioned fibres to the superior rectus and levator muscles, and the lid is able to follow the eyeball. Under certain circumstances, notably those found in certain cases of exophthalmic goitre, this relationship is disturbed, and the upper lid is mechanically not in condition to follow the eyeball.

Stellwag's Sign consists of diminished frequency of and imperfect winking. A rapid succession of imperfect winks may be followed by a long pause without winking. The winking is imperfect in that the lids do not meet. The insufficiency is in part due to lessened reflex irritability of the cornea and retina. Sattler considers the lesion one of the reflex centres governing the retina, the cornea, and the conjunctiva. Swanzy thinks the imperfect winking is probably due to insufficiency of the orbicularis rather than to overaction of the levator.

Infrequent nictitation may be found in hystero-epilepsy. It was also noted by Savage in a twenty-five-year-old woman who was otherwise healthy. He could examine the fundus with the ophthalmoscope for half an hour without the patient winking.

On account of the imperfect closing of the lids, the lower portion of the cornea may suffer ulceration or pannus formation.

Dalrymple's Sign.—This is a retraction of the upper lid, with consequent widening of the palpebral fissure. It gives to the patient the characteristic stare and look of apprehension. Other conditions producing

a similar widening of the palpebral fissure are orbital tumor (mechanically), stimulation of the cervical sympathetic, cocain (probably by action on the sympathetic), hysteria (occurring in women after childbirth), tetanus (spasm of the occipito-frontalis muscle), and complete amaurosis.

Another phenomenon, which is, however, not characteristic of exophthalmic goitre, is tremor of the upper lid when an effort is made gently to close the eye (Rosenbach's phenomenon). It is due to an antagonism or want of harmonious action between the palpebral portion of the obicularis and the levator muscle. Joffroy has noticed that when the patient holds the head down and attempts to look up without raising the head, the forehead remains smooth. Normally under such circumstances it should wrinkle. Periodic edema of the eyelids has been noted in a number of cases, and in one case three years before the disease declared itself (Wilbrand and Saenger). Discoloration of the skin, similar to that found in Addison's disease, has been noticed in a few cases (Story).

Falling of the eyelashes and eyebrows occasionally occurs in the beginning or in the course of the disease. Sattler attributes it to trophic disturbance.

Conjunctivitis and lacrimation are common in cases with marked exophthalmos. The conjunctivitis is probably due to diminished sensibility of the conjunctiva and imperfect nictitation. The lacrimation and epiphora are thought by Berger to be due to stimulation of the sympathetic nerve, causing increased secretion from the lacrimal gland. Schmidt-Rimpler, on the other hand, considers these symptoms to be the result of mechanical irritation of the conjunctiva, which, on account of the proptosis and widened palpebral fissure, is more exposed than is normally the case. Another element in the causation is displacement of the puncta lacrimalia. The cornea suffers keratitis e lagophthalmo; opacities may appear on the lower portion, followed possibly by ulceration, or the unprotected portion of the cornea may become covered with pannus. One or both eyes may be destroyed. Mooren and Spalding have recorded cases in which enucleation was necessary on account of purulent chorioiditis.

The exophthalmos is probably due to increase of the retrobulbar orbital contents. That there is a temporary increase in the amount of orbital fat has been clearly demonstrated; also there is an increased amount of blood in the orbital vessels.

In accordance with the theory of sympathetic irritation, contraction of Müller's orbital muscle, which covers the spheno-maxillary fissure, as well as contraction of other smooth muscle-fibres in the orbit, has been advanced as a causative factor of the exophthalmos. After death the exophthalmos in part disappears, which, of course, favors the theory of muscular spasm and increased blood-supply. Knies states that dilated pupils as well as unequal pupils, with retained reflex activity, are frequently found. This he attributes to sympathetic irritation.

Contrary to what might be expected, fundus changes in exophthalmic

goitre are generally wanting, or, if present, are neither pronounced nor characteristic. In some cases the arteries are enlarged from vasomotor paralysis. They may equal the veins in calibre. Spontaneous arterial pulsation was found by Becker six times in seven cases. The veins may be tortuous. Very rarely optic neuritis and optic-nerve atrophy are found.

It should be remembered that exophthalmic goitre occurs often coincidently with other diseases of the nervous system, or constitutional diseases, such as hysteria, multiple sclerosis, bulbar paralysis, diabetes mellitus, or diabetes insipidus. Care must therefore be observed not to attribute to exophthalmic goitre changes which might be due to other diseases.

NATURE OF THE DISEASE.—The pathology of exophthalmic goitre is as yet not known with certainty. For a full discussion of the various views on this subject works on nervous diseases and internal medicine must be consulted. Suffice it here to say that there are two chief theories, each of which is warmly supported by many competent observers. It seems, however, that the greater amount of evidence is in favor of the disease being one primarily of the cervical sympathetic. Gordon says that the theory based on the influence of the sympathetic nerves covers all cases without exception. The other theory is based on an increased thyroid secretion (hyperthyroidization) as well as a perversion of the secretion. This is held to be the primary cause, the other changes being produced secondarily.

PROGNOSIS.—In cases of pure Graves's disease this may be said to be good. As mentioned above, however, there is a great tendency for Graves's disease to occur coincidently with other diseases, in which the prognosis is not so favorable. Occurring in highly nervous and debilitated individuals, the prognosis is not so favorable as when occurring in individuals who have previously enjoyed robust health, and are not members of that large class of neurotics. Williams, of Manchester, in a study of 24 cases found 6 to be fatal, complete recovery in 7, improvement in 7, the condition unchanged in 3, and 1 case in which the patient was following his occupation, but the exact condition was unknown. The cases not dead or recovered were observed for a period of five years.

TREATMENT.—Works on internal medicine and neurology must be consulted for the general treatment of exophthalmic goitre. At the present time surgical measures for the relief of this affection are being actively discussed and tried. Such are simple exposure of the thyroid gland (exothyropexy), resection of the cervical sympathetic ganglia, ligation of three of the thyroid arteries, and partial extirpation of the gland. The ocular complications must be met as they arise. If the protrusion of the eyeball is sufficient to threaten the cornea from exposure, the palpebral fissure may be reduced in length by tarsorrhaphy. If more radical measures are necessary, the edges of the lids may be denuded and the two united by stitches in their entire extent, thus obliterating the palpebral fissure. The fissure may at the proper time be re-formed. Protection of the cornea is the important point to be kept constantly in mind.

WOUNDS AND INJURIES OF THE ORBIT.

Under this heading we have for consideration simple contusion of the periorbital tissues (black eye), contused and incised wounds around the orbital margin, punctured and penetrating wounds of the orbital tissues, dislocation of the eyeball, fracture of the orbital walls, traumatic enophthalmos, and the entrance into the orbit of foreign bodies.

Contusion of the periorbital tissues (black eye) results from blows at the entrance of the orbit, and in this country and in England is often regarded as evidence of a preceding fistic encounter. Uncomplicated, the symptoms are those of any ordinary contusion. Blood is extravasated within the tissues of the lids and under the conjunctiva, causing the characteristic discoloration. The eyelids are swollen, often to the extent of complete closure. The eyeball suffers in proportion to its prominence and the amount of violence sustained. Emphysema indicates a fracture establishing communication with air-spaces, the ethmoid usually being fractured in these cases, thus opening the ethmoidal cells.

The prognosis is good. The treatment aims at absorption of extravasated blood. To this end hot fomentations and pressure are of use. Recently a mixture of potter's clay and glycerin, under the copyrighted name of antiphlogistin, has been used with good results.

Injuries to the Orbital Margin.—Considering the sharpness and rigidity of the orbital margin, it is not surprising that rather moderate blows here frequently cause wounds which, though contused in mode of production, often have the appearance of being incised. Owing to their contused nature, they have more or less of a tendency to suppurate. They should be carefully cleansed, stitched, and protected antiseptically.

A much discussed question in former years was the occurrence of blindness following blows at the margin of the orbit, there being no demonstrable injury to the eyeball itself. That this has occurred many times is well known. The cause is to be sought in injury to the optic nerve, either from fracture in the neighborhood of the optic foramen or from hemorrhage into the orbit destroying the nerve by pressure. The supra-orbital and infra-orbital nerves are often injured by blows in this situation, causing anesthesia of the parts supplied, but there is no known association between these nerves and the optic nerve, as some writers have endeavored to prove.

Wounds of the Soft Tissues of the Orbit are usually punctured in character. They are caused, as a rule, by sticks, bayonets, umbrella ferrules, swords, etc., being thrust into the orbit, or by missiles of almost every kind striking at the orbital entrance. The bones may or may not be fractured; the periosteum may be injured or not, according to the depth of penetration, and the supra-orbital and infra-orbital nerves may be lacerated or severed. The ocular muscles may be cut or completely detached, the eyeball ruptured, the optic nerve injured, or the eyeball dislocated. The symptoms and prognosis correspond with the extent of the injury.

TREATMENT must meet the exigencies of the case, and must accord with general surgical principles. If the muscles are severed an attempt should be made to find the ends and unite them. If the eyeball is ruptured it should be saved, if possible, by repairing the rent and by careful antiseptic dressing. If such efforts are not warranted, it should be enucleated, or better still eviscerated and an artificial vitreous implanted. Probing of the wound must be done only with great care and scrupulous cleanliness.

Luxation and Avulsion of the Globe.—As Barck has said, it is necessary to make a distinction between these terms. *Luxation* means that the eyeball lies in front of the lids, which close spasmodically behind it. The optic nerve and muscles are stretched, but their connection with the globe and with the other orbital structures is maintained. *Avulsion* indicates that, owing to rupture of the optic nerve and the severance of the majority or all of the extrinsic muscles, the connection is broken.

Luxation may occur spontaneously, as in some cases of exophthalmic goitre, and in persons with abnormally shallow orbits and prominent eye-

Fig. 365.—Avulsion of the eyeball. (BARCK.)

balls. It may also occur when, after the introduction of an eye speculum, the patient struggles, cries, or coughs. Dépoutol saw a young man with an opisthognatous face who could dislocate his eyeball by blowing the nose violently. Generally, however, luxation results either from the impact of a large blunt foreign body; from self-inflicted injury produced by the insane with their fingers; or from the barbarous practice of "gouging," which is said to be common among the natives of Uganda, Africa. In a few instances luxation has occurred in difficult instrumental deliveries.

Avulsion is produced in this manner: the missile causative of luxation also acts as a lever, with sufficient energy to sever the optic nerve and lacerate the muscles. The unyielding orbital margin acts as a fulcrum. Avulsion has been caused by falls upon blunt projecting bodies. Thus, it has resulted from a fall upon keys which were in their locks, from the impact of a boar's tusk, etc.

DIAGNOSIS.—Inspection will at once reveal the true condition.

PROGNOSIS.—In luxation the eyeball may or may not be ruptured. If

torn, the case will probably require enucleation. If the globe is simply luxated the prognosis is generally favorable, although severe damage may have been done to the optic nerve, retina, chorioid, and lens. In avulsion the eye is lost.

TREATMENT.—This consists in the earliest possible replacement of the eyeball within the orbit. It may be easily accomplished by separating the lids and gently pushing the globe backward. If it is attended with great difficulty, owing to the swelling of the lids and their tight constriction, retractors should be passed under the lids. In the event of failure, the outer canthus must be cut, the lids replaced, and the wound stitched. After reduction, rest and iced compresses are indicated, after which a compress bandage should be worn for some time. If the eyeball is ruptured the indications for treatment are such as are described elsewhere for this condition. If a case of avulsion is seen early and some of the muscles are still attached, an attempt may be made to stitch the globe into place with the hope of saving it for cosmetic reasons. If there is cause to believe that the germs of infection have been carried into the orbit, an enucleation should be made.

Fracture of the Orbital Bones.—Orbital fracture is not rare after head injuries, and it is probable that, were we able to examine more exactly the walls of the orbit, many cases of fracture would be found which at the present time it would be impossible to diagnose. Many of the bones are exceedingly thin and delicate, and are placed between bones of much greater density. It is doubtful if they are thus better protected or are more liable to injury; a box car between two Pullman coaches in a railroad collision is always broken to splinters.

The roof, the floor, and the inner wall of the orbit are all thin; the margin, the external wall, and the apex are quite dense and strong. Fracture involving the roof renders the patient liable to meningitis, and these cases frequently end fatally. Fracture of the floor establishes a communication with the antrum, and often injures the infra-orbital nerve, while fracture of the inner wall opens the ethmoid cells. When air-spaces are broken into, emphysema of the orbital cellular tissue is a symptom. Fracture at the apex, if through the optic foramen, almost to a certainty destroys sight; if through the sphenoidal fissure, the third, the fourth, the ophthalmic division of the fifth, and the sixth nerve are liable to injury, as well as are the ophthalmic and some smaller veins.

Fracture of the outer orbital wall is the most easily diagnosed with certainty, and is attended with results generally less serious than when the fracture is elsewhere—with the exception of fracture limited to the margin. Small fragments of the orbital margin are not infrequently broken off, and cause very little trouble, becoming reattached, leaving only a slight inequality or deformity. It is probable, however, that in many of these cases there is a fissured fracture running backward into the orbit. The most dangerous fractures are those of the roof.

The investigations of von Hölder are instructive. He examined the bones in 124 cases of fracture of the skull, giving the following summary: Of the 124 cases, 84 were fractures at the base; and, of these, the fracture in 79 cases extended into the orbital roof. In 63 of the 84 cases he found a fissure or fracture running through the optic canal, always through the upper wall of the canal, and in some of the cases through the inner wall also. In 42 cases there was hemorrhage into the optic-nerve sheath, but never unless the canal was fractured.

These figures throw much light on that rather large class of cases in which blindness follows head injuries. Such blindness may come on at the time of injury and remain permanent; or, coming on at the time of injury, vision may ultimately be in part or wholly regained; or impairment of vision may appear at a time subsequent to the injury. The cases of immediate permanent blindness have probably sustained laceration or even complete section of the optic nerve, by bone fragments derived from a fractured optic canal. Subsequent improvement of vision would indicate the removal of the immediate cause of blindness. Such would be, for instance, relief of pressure upon the optic nerve. Pressure of a lesser degree, acting for a longer time, might cause a slow deterioration of the optic nerve, ending in partial or complete blindness at a time more or less remote from that of the injury.

Some of these cases may show little or no ophthalmoscopic evidence of the injury at the time of its receipt. After extensive hemorrhage into the sheath of the optic nerve, the ophthalmoscope would show full veins and reduced arteries, fresh hemorrhages into the retina or even into the vitreous, blurring of the disc-margins, and retinal edema. The later appearances are those of neuroretinitis and optic-nerve atrophy.

Cerebral symptoms, due to intracranial hemorrhage and inflammation, are to be expected when the roof of the orbit has been broken through, and it is worthy of note that they may be delayed a long time. In one case referred to by Noyes, cerebral symptoms appeared forty days after injury by a foreign body. In a series of 52 cases of perforating wound of the orbital roof collected by Berlin, 41 died and 11 recovered. Of the 11 recoveries, 3 were hemiplegic, 1 had persistent headache, and 1 became an imbecile. A symptom of importance, although not constant, when the cribriform plate of the ethmoid is broken, is the dropping of a clear fluid from the nostril on the side of the injury. Bleeding from the nose is evidence of ethmoidal fracture; hemorrhage into the mouth has usually come through the antrum, and points to a fracture in the floor of the orbit. Hemorrhage into the orbit in some degree is a natural consequence of almost all orbital fractures, the former being in many cases only a symptom of the latter. Exophthalmos will depend upon the amount of hemorrhage. Enophthalmos may follow fracture at certain situations in the orbit, but, owing to the still unsettled views as to the pathogenesis of this condition, it has been considered separately (page 640).

The symptomatology of orbital fracture is always the same, however caused. The mode of production, however, whether by a blow, a thrust from a bayonet, sword, umbrella ferrule, or whether from a gunshot, is of considerable clinical value in showing the direction of immediate impact and in giving an idea as to the extent of injury. A punctured wound from below upward is much more to be feared than one from above downward or one directed toward the temporal wall. Gunshot wounds are here, as elsewhere, very uncertain; the balls glance, and may take any or many directions. They are a little like bolts of lightning, deciding at the time which way they will go next. Furthermore, punctured and penetrating wounds may be probed and valuable information may be gained thereby. Probing, however, must be done with great care, cleanliness, and judgment. A probe in the hands of an unskillful surgeon may be a source of danger not exceeded even by the instrument causing the original injury.

TREATMENT.—The treatment of fracture of the orbital bones and of the complications above described is that which general surgical principles dictate. The surgeon must keep before his mind always the very many contingencies, both near and remote, which may arise, and examine repeatedly along every available line of investigation for signs which will indicate what is occurring within. At the same time he should avoid being meddlesome, and, if through a penetrating wound he has with a probe once demonstrated to his satisfaction the existence of a fracture, he should be content to believe that any further probing can be only harmful. If the injury has been inflicted by some tool or instrument, an examination of the tool or instrument should, if possible, be made, to determine if any portion of it is missing which may have remained in the orbit. Pus must be evacuated, injured structures (so far as possible) repaired, and function preserved.

Foreign Bodies in the Orbit.—Foreign bodies may enter the orbit and remain for a long time undetected, or they may at once manifest themselves in ways depending upon the amount of damage done. Small bodies—such as shot—are often very difficult or impossible to locate, and in many cases may remain within the orbital tissues, become encapsulated, and cause no trouble.

The path of entrance of a foreign body into the orbit is, as a rule, sinuous, and therefore difficult satisfactorily to probe. As explained by Noyes, the body entering the orbit reaches the fasciæ and forcibly drags the eyeball around toward the side of entrance; when the body has come to rest, the eyeball returns to its normal position, thus making the path sinuous and corresponding not at all to the direction of entrance.

Fracture of the orbital walls or serious injury to the intra-orbital structures is more frequently found than not, if the foreign body enters with considerable force. Any foreign body which can be located should be at once removed. The locating of many foreign bodies has been much facilitated by the recent ingenious application of the Roentgen rays. Ex-

cellent methods have been devised by Sweet and Leonard, of Philadelphia, and by Davidson, of London.

If, after an injury which is apt to result in a foreign body being left within the orbit, a suppurating sinus remains, it is very good evidence that the body is still there. After all injuries of this kind, the patient should be carefully watched for a number of days for any signs of bone disturbance or pus-formation. Cases are on record of foreign bodies passing through the roof of the orbit into the brain. They have given no adequate symptoms at the time, but at a later period have caused death from brain-abscess. The patient should be carefully questioned as to the manner of injury, and all objects implicated in the propulsion of the offending body should be examined for broken fragments. Such a procedure is often useful in dealing with mechanics who are injured while at work.

OPERATIONS ON THE ORBIT.

Orbital operations are demanded for the removal of tumors, cysts, and foreign bodies; for the repair of damage caused by the entrance of foreign bodies into the orbit; for the evacuation of abscess-cavities, the removal of necrosed bone; for resection of the optic nerve; and frequently for the relief of diseased processes in the adjacent cavities. Operations for prosthesis in cicatricial orbits have been described in Chapter XVIII.

Whatever operation is attempted in this region, the principles of modern surgery, regarding the preparation of the patient and that of the instruments, should prevail throughout. The proximity of the brain and the ready ways of access thereto should ever keep before the operator's mind the possibility of subsequent meningitis.

Removal of Tumors.—In dealing with intra-orbital cysts and benign tumors the prime object should be to remove them without destroying the eyeball or the optic nerve. If the tumor be malignant, it is generally advisable to remove the eyeball and in some cases to clean out the entire orbit (exenteration). Knapp always approaches these cases in an exploratory manner, being fully prepared at once to continue the operation to meet the exigencies of the case. This is a wise procedure, for the extent and attachments, as well as the character, of many growths cannot be determined until they are brought to view.

Growths situated at or presenting near the orbital entrance can be exposed by making a sufficient incision concentric with the orbital margin, separating the tissues with a blunt instrument, and displacing the eyeball and adnexa upward, downward, inward, or outward as the case requires. If within the muscle-cone, access is obtained by exposing the internal rectus muscle, passing a silk thread through it near its insertion, then detaching the muscle at its insertion, and with the thread drawing and anchoring the muscle away from the eyeball. Strong rotation of the eyeball outward will open up the space within the cone. The growth may then be stripped from

its attachments as far as possible and removed. This done, the rectus muscle is reattached at its original insertion in the sclera. In this manner the optic nerve is also to be exposed, and, if resection is required, one hook is passed around it near the globe and a second hook worked backward as far as necessary, the intervening nerve being the portion for resection.

Krönlein's Operation, practiced first in 1887, is for the exposure of the deep portions of the orbit. It is a difficult and tedious operation, but if properly done deep-seated growths or foreign bodies can be much more readily removed than by working from the orbital entrance. The operation consists of a temporary osteoplastic resection of the outer orbital wall. An incision starting from the temple is made to pass through the upper, outer orbital margin, then, curving within the margin (to the nasal or median side), carried backward, again crossing the orbital margin a little below its horizontal meridian and ending in the temple above the zygomatic bone. The incision in its two crossings of the orbital margin, and the

A-B, Skin incision. *A-B*, Bone resection.

Fig. 366.—Krönlein's operation.

included curve is carried down to the bone. The periosteum of the orbital surface of the outer wall is then detached as far back as the anterior end of the spheno-maxillary fissure. By chiseling from the upper end of the denuded margin obliquely downward to the spheno-maxillary fissure, and from the lower end of the margin to the anterior end of the fissure, or by use of a dental engine and circular saw, a wedge-shaped piece of bone is loosened. This, with its skin and muscle attachments, may now be drawn outward, thus permitting of ready access to the deeper portions of the orbit. The periosteum must be slit before the intra-orbital contents can be reached. When the work within the orbit has been completed, hemorrhage must be checked, after which the resected bone is pushed into place, the periosteum is united with catgut sutures, and the skin-flaps are united with silk. The after-treatment consists of a compress bandage and rest in bed until healing is well under way.

Exenteration of the Orbit consists in removal of the orbital contents in their entirety. Its indication is usually malignant disease which is no longer isolated, but involves many or all of the orbital structures. If the entire periosteum is healthy, it may be allowed to remain (partial exenteration); if diseased, it must be stripped off (total exenteration).

The first step of the operation is to divide the outer canthus; an incision is then made through the conjunctiva of the everted lower lid down to and along the lower margin of the orbit. A similar incision is carried along the outer, upper, and finally along the inner margin. If the exenteration is to be total, the periosteum is divided along the margin and is stripped from the bone back to the apex; the apical mass of tissue is then divided with heavy curved scissors or with a sharp curette. The orbital contents are then removed enveloped in the periosteum.

If the exenteration is to be partial, the preliminary steps of the operation are the same as for total exenteration; then, instead of dividing the

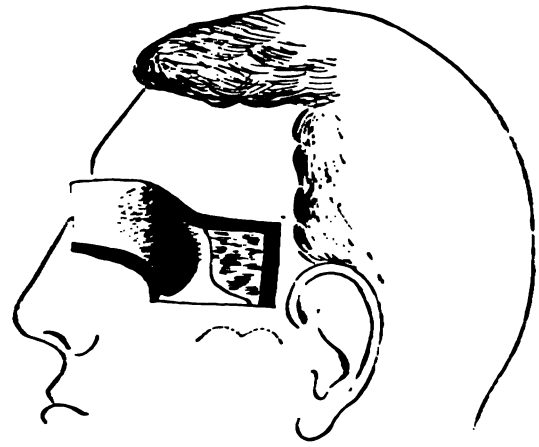


Fig. 367.—Modification of Krönlein's operation. (Parinaud and Roche.)

periosteum, the soft tissues are separated from it with scissors and the apex is cut or curetted as above. In either case the eyeball may first be enucleated in the regular way or not, as desired. Hemorrhage in this operation is generally copious, but is easily controlled by packing aided by hot water. The dressing consists of sterile gauze packing, which should remain undisturbed for two or three days before repressing in the same manner.

In stripping the orbital cone of the periosteum the thinness of the bones at certain points should not be forgotten, and great care should be exercised especially at the roof. If the lids are not involved in cases of malignant disease, it often becomes necessary to remove them along with the orbital contents. The resulting deformity is, of course, much increased. The entire orbit may be closed in or excluded by the grafting of skin from the forehead, temple, and cheek. The deformity is still considerable, but far less revolting than when the orbit is uncovered.

CHAPTER XX.

ANOMALIES OF THE MUSCULAR APPARATUS.

By WILLIAM ZENTMAYER, M.D., of Philadelphia,

One of the Attending Surgeons to Wills Eye Hospital and Ophthalmic Surgeon to St. Mary's Hospital.

In order to understand the phenomena resulting from disturbed action of the orbital muscles it is essential to have a clear conception of the individual and associated action of these muscles under normal conditions.

The movements of the eyeball are effected by means of three pairs of

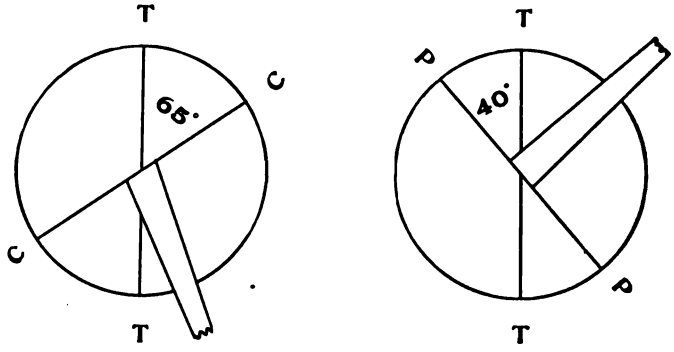


Fig. 368.—Scheme of the axes of rotation of the ocular muscles.

The figure at the left: *T-T*, antero-posterior axis of the eyeball; *C-C*, axis of rotation of the superior and inferior recti of the left eye. The figure at the right: *P-P*, axis of rotation of the superior and inferior obliques of the left eye.

muscles, which move the eye about the centre of rotation. Each muscle of a pair is the antagonist of the other.

All movements of the eyeballs are to be considered as starting with the eyes in the *primary position*—that is, with the head erect and the eyes directed straight forward to a distant point on the visual plane.

Action of the Muscles.—The muscular plane of the *internal* and *external recti* corresponding to the horizontal plane of the eyeball, these muscles move the globe respectively directly inward and outward about a vertical axis.

Owing to the attachment of the *superior and inferior recti* being anteriorly and templeward relative to their point of origin, the muscular plane of each is inclined to the antero-posterior axis of the globe, forming with it an angle of about 25 degrees, and consequently the muscles move the eye about an axis directed from before and nasalward to behind and templeward, forming with the antero-posterior axis an angle of about

65 degrees. The superior rectus, therefore, does more than move the eye upward, associating with this principal action an inward movement and a partial rotation of the cornea (torsion), so that the vertical meridian becomes inclined by its upper extremity toward the nose; while the inferior rectus moves the eyeball downward and inward and inclines the vertical meridian of the cornea outward by its upper extremity. The supra- and infra- ducting power of these muscles is greatest when the eyeball is turned outward, as then the horizontal axis of the globe coincides most nearly with the axis of rotation of these muscles; while the torsion action is most pronounced when the eyeball is turned inward, as in this position the antero-posterior axis of the globe coincides most nearly with the axis of rotation of these muscles.

The attachment of the *obliques* being posteriorly and templeward relative to the point of mechanical origin, the muscular plane is in-

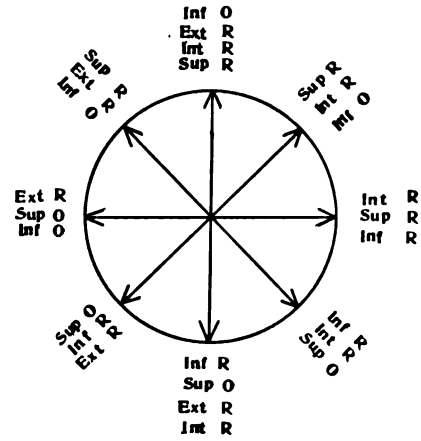


Fig. 369.—Muscles associated in moving the eyeballs in the directions indicated by the arrows.

clined to the antero-posterior diameter of the globe, forming with it an angle of about 50 degrees. Consequently these muscles move the eyeball about an axis directed from anteriorly and templeward to posteriorly and nasalward, forming with the antero-posterior axis of the eyeball an angle of about 40 degrees. The *superior oblique,* therefore, turns the eyeball downward and outward and inclines the vertical meridian of the cornea inward by its upper extremity. The *inferior oblique* turns the eyeball upward and outward and inclines the vertical meridian of the cornea outward by its upper extremity. The supra- and infra- ducting power of the obliques is greatest when the eyeball is turned in, as in that position the horizontal axis of rotation of the globe coincides most nearly with the axis of rotation of the obliques, and the torsion power is greatest when the eyeball is turned outward, as in this position the antero-posterior axis of the globe agrees most nearly with the axis of rotation of these muscles.

As in accordance with the *law of Listing* all rotations of the eyeball must be "about axes in a vertical plane passing through the centre of motion of the eye perpendicular to the visual line in its primary position" (Maddox), it follows that the independent physiologic action of any muscle which would revolve the eye about an axis inclined from either the vertical or horizontal is impossible; consequently all movements of the globe other than the lateral must be accomplished by the co-ordinate action of such muscles as would have the resultant axis in this plane.

The outward movement of the eyeball is effected by the ***external rectus,*** aided by the *superior and inferior obliques.*

The *inward* movement of the eyeball is effected by the ***internal rectus,*** aided by the *superior and inferior recti.*

The *upward* movement of the eyeball is effected by the ***superior rectus*** and *inferior oblique* acting together, the *lateral recti* serving to guide the eyeball.

The *downward* movement of the eyeball is effected by the ***inferior rectus*** and the *superior oblique* acting together, the *lateral recti* serving to guide the eyeball.

In the upward movement the inward rotation and the slight inward inclination of the globe which would result from the action of the rectus alone are opposed by the counter-action of the ***inferior oblique.***

In the downward movement the inward rotation and the slight outward inclination of the globe which would result from the action of the rectus alone are opposed by the counter-action of the ***superior oblique.*** The muscles associated in turning the eye into oblique positions may be learned from Fig. 369. The eye should be capable of an upward rotation of at least 33 degrees, downward of at least 50 degrees, nasalward and templeward from 48 to 53 degrees (Stevens).

Binocular Movements.—The turning of the eyeballs to the right is accomplished by the associated action of the *external rectus* and the *obliques* of the right eye with the *internal rectus* and the *superior* and *inferior recti* of the left eye; to the left, by the associated action of the external rectus and the *superior* and *inferior obliques* of the left eye with the internal rectus and the *superior* and *inferior recti* of the right eye.

The muscles associated in oblique conjugate deviations are:—

Up and to the right	R. E.	*Superior rectus, inferior oblique,* and *external rectus.*
	L. E.	*Superior rectus, inferior oblique,* and *internal rectus.*
Up and to the left	R. E.	*Superior rectus, inferior oblique,* and *internal rectus.*
	L. E.	*Superior rectus, inferior oblique,* and *external rectus.*
Down and to the right	R. E.	*Inferior rectus, superior oblique,* and *external rectus.*
	L. E.	*Inferior rectus, superior oblique,* and *internal rectus.*

Down and to the left	R. E.	*Inferior rectus, superior oblique,* and *internal rectus.*
	L. E.	*Inferior rectus, superior oblique,* and *external rectus.*

Convergence of the visual lines results from the associated action of the *internal recti* of both eyes.

General Symptomatology.—When, from any cause, one of the extra-ocular muscles fails to act in association with its physiologic fellow (associated antagonist—von Graefe) of the other eye, disturbed binocular vision results, varying from a mere confusion or blurring of the outlines of objects to actual diplopia, provided that binocular vision previously existed, that the false image falls upon the retina, and that there is no associated paralysis of the palpebral levator muscle.

DIPLOPIA (double vision) results from the fact that images for both eyes of the same external object fall upon dissimilar points of the two retinæ, and are in consequence projected to different parts of the visual field.

Primary Deviation is the deviation of the affected eye when the sound eye fixes. It is often appreciable only when the eyes are turned somewhat in the direction of the action of the affected muscle.

Secondary Deviation is the deviation which the sound eye undergoes when the affected eye is made to fix. It is always greater than the primary deviation, because the excessive innervation which is necessary to cause the affected eye to fix is transmitted also to the sound associated muscle of the fellow-eye, and produces a corresponding excessive excursion of the eye in the direction of the action of this muscle. Example: With a paralysis of the external rectus of the right eye the object to be fixed is held at a distance of about 40 centimetres in front and slightly turned to the right side, and the patient is commanded to look sharply at it, the other eye being covered by a card. The cover is now transferred to the affected eye, when, in order to fix the object, the sound eye will be seen to make a large outward excursion, which is evidence that it must have been turned in excessively while under cover. In other words, the internal rectus muscle of the sound eye was receiving innervation in excess of the amount required to keep the visual line directed to the object.

Projection of the False Image.—The patient refers the false image to that position in space which (in the normally directed eye) an object would hold having its image formed on the point of the retina now occupied by the false image. The exact position of the projected image can be determined by giving to the eye its normal direction (which would be accomplished by action of the affected muscle) and projecting a line from this retinal point through the centre of rotation of the eye into space. Example: With a paralysis of the external rectus muscle, the internal rectus draws the eyeball inward, so that the image of an object fixed by the fellow-eye will, in the affected eye, fall to the inner side of the fovea. Now, this point of the retina, under normal conditions, receives the image of an object situ-

ated to the temporal side, and it will be in that locality that we will find that the patient refers the false image.

Limitation of the Movements of the Globe is always in the direction of the action of the affected muscle, and varies from abolition to a degree so slight as to be detectable only by accurate determination of the field of fixation. With both eyes open, the patient is directed to follow, with the eyes, the movement of the tip of a pencil, or like object, which is carried in a systematic manner through the principal meridians. The ability or inability of the two eyes to follow in an equal degree is determined by comparing the distances between the margin of the cornea and the edges of the lids, in the upward and downward movements, and the canthi in the lateral

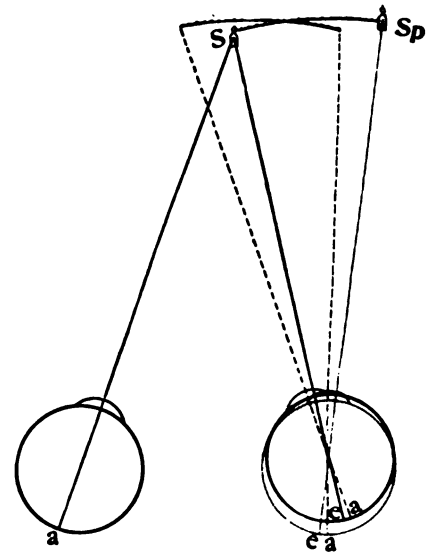


Fig. 370.—Illustrating the projection of the false image.

The right eye is convergent; hence the diplopia is homonymous. The red line represents the eye restored to its normal position, showing that the retinal point, *c*, which received the image when the eye was deviated, would receive the image of an object situated at *Sp*, when the eye is in the normal position. Hence it is to this position in space that the patient refers the false image.

movements in the two eyes; or by comparing the position of the corneal images of some distant object when the eyes are moved in the different directions. The *field of fixation* is determined in the same manner as is the visual field, except that the patient is directed to *follow* with the affected eye the test-object (which may consist of a small printed letter) as it is carried outward along the arc of the perimeter and make known when he can no longer decipher it. This method is of especial value where the defect is slight or where more than one muscle is affected. Example: In paralysis of the inferior oblique the field of fixation would show contraction above and to the outside. Stevens has designed an instrument, which he terms a tropometer, for measuring the rotation of the eyes in all directions. It con-

sists of a telescope in which an aërial image of the cornea is formed near the eyepiece (see page 110).

False Muscular Projection arises from disturbed relation between innervation and muscular action, so that a false, exaggerated conception of the direction of the eye is obtained, because of the amount of innervation required to bring the visual axis to bear on the object. It manifests itself in an inability properly to judge of the location of objects. Example: In paralysis of the internal rectus muscle of the left eye, if the patient be asked to place his finger on an object held slightly toward the right side, he will direct his finger farther to that side than the object is situated, because the amount of innervation required would, under normal conditions, have directed the visual line to that point.

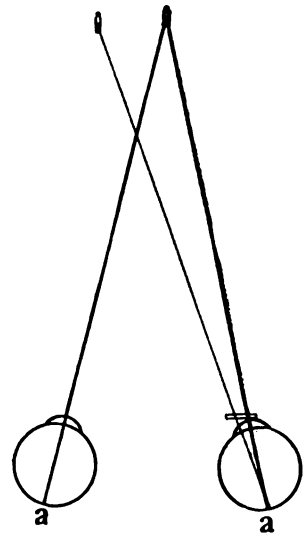


Fig. 371.—Paralysis of the internal rectus of the right eye, producing crossed diplopia.

The red glass is before the right eye.

Vertigo results from the diplopia and from defective orientation—that is, inability to judge correctly of the relative location of objects in space.

Compensatory Turning of the Head to Avoid Diplopia.—The explanation of this symptom is that the patient, being unable to turn the affected eye, turns the head so as to bring the visual line to bear upon the object fixed by the sound eye—that is, the head is turned in the direction of the defective movement of the eye. Example: In paralysis of the internal rectus of the left eye the head is carried to the right.

Method of Examination.—The examination for the determination of the characteristics of the diplopia should be carried on in a dark-room with the patient seated. The greatest care must be taken that the head is held erect and motionless, lest by moving the head the patient compensate for the deviation of the eye. In order better to differentiate the images a

red glass should be held before one eye. The use of a frame is to be avoided, as in extreme excursions of the eyeball it may obstruct the vision and lead to false conditions. The examiner should stand with a lighted candle about three metres in front of the patient and direct him to follow the movements of the light with his eyes, and note the relation of the two images as the candle is carried along certain meridians through which the eye may be normally moved. The room should be large enough to prevent projection of the images upon the side-walls. In some instances the image falls far out on the periphery of the retina, or perhaps on the papilla (Wallace) and is not recognized. In such a case a prism, so placed as to bring the image on to a sensitive part of the retina, will render the diplopia manifest. By this method

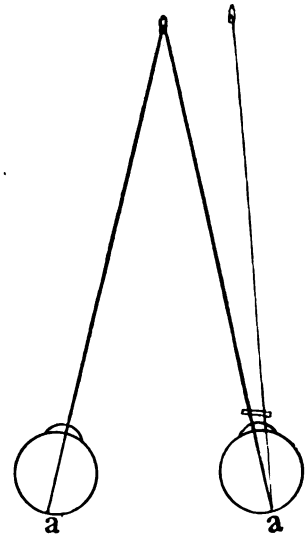


Fig. 372.—Paralysis of the external rectus of the right eye, producing homonymous diplopia.

The red glass is before the right eye.

no great difficulty will be experienced in arriving at a proper diagnosis if but one muscle be paralyzed. Should it be evident that two or more muscles are affected, a prism will sometimes be found of service in eliminating the diplopia resulting from the paralysis of one muscle, and thus will simplify the further study of the images. As a rule, however, in these cases it will be found necessary to mark upon a blackboard the relation of the images as they appear in various parts of the field of fixation.

General Principles.—The distance between the images increases when the light is carried in the direction of the action of the affected muscle. The fainter image belongs to the affected eye. When the images are *crossed*,—that is, when the red image is seen to the opposite side of the normal image, from the eye before which the red glass has been placed,—an adductor muscle is affected, and one of the vertical straight muscles, if the

images are not on the same horizontal plane. Should the red image be seen on that side of the normal light corresponding to the eye before which the red glass has been placed, the images are said to be *homonymous*, and it indicates an affection of one of the abductor muscles, and of one of the obliques if the images are not on the same horizontal plane.

A greater separation of the images occurs when the eyes are directed to the image belonging to the affected eye.

The false image has the position and the inclination which the affected muscle gives to the eyeball when acting normally. The remembrance of this principle and of the facts necessary to apply it is a *sine qua non* for the solution of the succeeding problems.

Special Symptomatology.—PARALYSIS OF THE EXTERNAL RECTUS (HORIZONTAL DIPLOPIA).—Images are upon the same plane, homonymous,

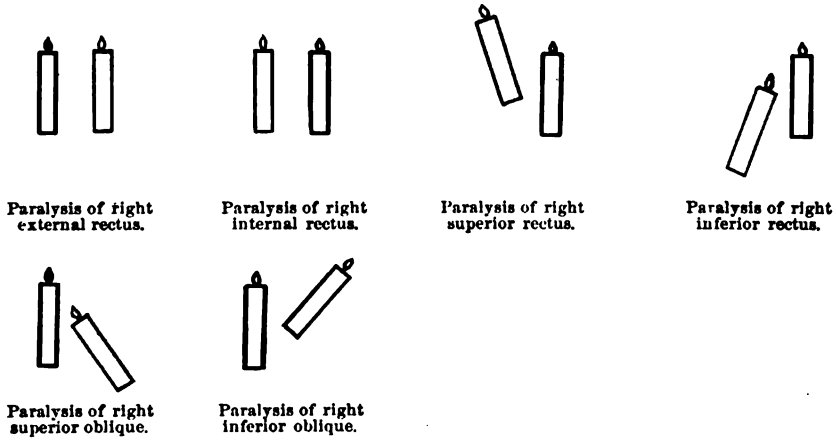

Paralysis of right external rectus.
Paralysis of right internal rectus.
Paralysis of right superior rectus.
Paralysis of right inferior rectus.
Paralysis of right superior oblique.
Paralysis of right inferior oblique.

Fig. 373.—Position of the images in ocular paralyses.

The true image is black, the false is red.

and parallel; separation increases in the direction of the affected side, and also in looking downward.

Primary deviation—inward.
Secondary deviation—inward.
Limitation of excursion—outward.
Field of fixation—contracted outward.
Inclination of head—turned toward the affected side.
False muscular projection—toward the affected side.

INTERNAL RECTUS (HORIZONTAL DIPLOPIA).—Images are upon the same plane, crossed and parallel; separation increases toward the sound side, and also in looking upward.

Primary deviation—outward.
Secondary deviation—outward.
Excursion—limited inward.
Field of fixation—contracted inward.
Inclination of head—turned toward the sound side.
False muscular projection—toward the sound side.

SUPERIOR RECTUS (DIPLOPIA IN THE UPPER FIELD).—Images are superposed (vertical diplopia) and slightly crossed; false image leans by upper extremity toward the sound side. Vertical separation increases in the upper field, and is greater up and out. Lateral separation increases toward the sound side, and the inclination is greatest toward the sound side.

Primary deviation—downward and slightly outward.
Secondary deviation—upward and toward the sound side.
Excursion—limited upward.
Field of fixation—contracted upward and inward.
Inclination of the head—turned up and toward the sound side, with a slight inclination toward the shoulder of the sound side.
False projection—up and toward the sound side.

INFERIOR RECTUS (DIPLOPIA IN THE LOWER FIELD).—Images are superposed (vertical diplopia) and slightly crossed; false image leans by its upper extremity toward the affected side. Vertical separation increases downward, and is greatest down and out. Lateral separation increases toward the sound side, and the inclination is greatest toward the sound side.

Primary deviation—upward and slightly outward.
Secondary deviation—downward and toward the sound side.
Excursions—limited downward, and downward and inward.
Field of fixation—contracted downward and inward.
Inclination of the head—downward and toward the sound side, with a slight inclination toward the shoulder of the affected side.
False projection—downward and to the sound side.

SUPERIOR OBLIQUE (DIPLOPIA IN THE LOWER FIELD).—Images are superposed (vertical diplopia) and slightly homonymous; false image leans by its upper extremity toward the sound side. The vertical separation increases downward and is greatest down and in. The lateral separation increases toward the affected side, and the inclination is greatest toward the affected side.

Primary deviation—upward and slightly inward.
Secondary deviation—downward and toward the affected side.
Excursions—limited downward, and downward and outward.
Field of fixation—contracted downward and outward.
Inclination of the head—downward and toward the affected side, with a slight inclination toward the shoulder of the sound side.
False projection—below and toward the affected side.

INFERIOR OBLIQUE (DIPLOPIA IN THE UPPER FIELD).—Images are superposed (vertical diplopia) and slightly homonymous; false image leans by its upper extremity toward the affected side. The vertical separation increases upward and is greatest up and in. The lateral separation increases toward the affected side, and the inclination is greatest toward the affected side.

Primary deviation—downward and inward.
Secondary deviation—upward and toward the affected side.
Excursions—limited upward, and upward and outward.
Field of fixation—contracted upward and outward.
Inclination of the head—held upward and turned slightly toward the affected side, with a slight inclination toward the shoulder of the affected side.
False muscular projection—above and toward the affected side.

Occasionally in paralysis of either the superior oblique or of the inferior rectus the false image appears closer to the patient than does the true one. This has been explained by the fact that the false image, which is the lower one, must appear closer to the patient if he projects them on to a horizontal plane as represented by the floor.

Crossed diplopia is occasionally met with in paralysis of either oblique. The explanation of this anomaly given by Mauthner is that a previously existing latent exophoria has become manifest.

PARALYSIS OF THE OCULOMOTOR NERVE.—When all the muscles supplied by the third nerve are affected, the following characteristic clinical picture will be observed: Ptosis with compensatory contraction of the occipito-frontalis muscle, outward and slightly downward deviation of the eye, loss of all movements of the globe except outward and outward and downward, fixedly semidilated pupil, and loss of accommodation. There is diplopia in all parts of the field except downward and outward.

EXAMPLE OF THE CLINICAL METHOD OF DETERMINATION.—Having arranged as directed in the paragraph on "Method of Examination" (page 659), with the red glass before the right eye, the patient will see two lights —one red and the other of the natural color. Inquiry brings out the fact that the lights stand one almost above the other, with the red one the higher and a little to the patient's left. This determines that either a levator or a depressor is affected, and, as the images are crossed, it must be one of the straight muscles. On carrying the candle into the upper field the patient states that the distance between the lights increases, from which we decide that the affected muscle is a levator, and of the right eye, as the image of that eye stands the higher. Should the patient fail to realize that the images do not stand one directly above the other, the diagnosis cannot at this stage be made, and the light must be carried into the superior lateral fields in order to accentuate the inclination of the false image. If the patient answers that this follows when the candle is carried toward the sound side, it renders possible a diagnosis of paralysis of the right superior rectus muscle.

Duane considers that paralysis of the obliques and of the superior and inferior recti can be diagnosed from the behavior of the vertical diplopia alone, and with this idea in view he has evolved a simple method for the determination of the muscle paralyzed. He divides the elevators into two groups: The first includes those having the greatest elevating effect when the eyes are turned to the right (the right superior rectus and the left inferior oblique). These he terms *right-hand elevators*. The second group

includes those having the greatest elevating effect when the eyes are turned to the left (the left superior rectus and the right inferior oblique)—*left-hand elevators.* The depressors are similarly grouped. The right-hand depressors are the right inferior rectus and the left superior oblique; and the left-hand depressors are the left inferior rectus and the right superior oblique. Duane summarizes as follows:—

"A vertical diplopia increasing not only up, but markedly up and to the right, means paralysis of a right-hand elevator (right superior rectus or left inferior oblique); and one that increases particularly up and to the left means paralysis of a left-hand elevator (right inferior oblique or left superior rectus). In either case a right diplopia (vertical diplopia, with the image of the right eye below) means paralysis of the muscle of the left eye, and left diplopia (vertical diplopia with the image of the left eye below) means paralysis of the muscle of the right eye.

"A vertical diplopia increasing down and to the right means paralysis of a right-hand depressor (right inferior rectus or left superior oblique), and vertical diplopia increasing as the eyes are carried down and to the left means paralysis of a left-hand depressor (right superior oblique or left inferior rectus). In either case if the image of the right eye is below (right diplopia) it is the muscle of the right eye that is paralyzed, and if the image of the left eye is below (left diplopia) it is the muscle of the left eye that is paralyzed."

Werner's scheme (Fig. 374) will be found of assistance in recalling the relation of the images in paralysis of the muscles. The broken lines represent the false images. The method of applying it may be illustrated by considering a paralysis of the right superior rectus. The diagram shows at once that the diplopia occurs in the upper field, and that the images are crossed, the false one standing the higher and being inclined by its upper extremity toward the sound side.

Etiology.—Clinically considered, the causes of ocular paralysis may be divided into two classes—*central* and *peripheral,* accordingly as the lesion lies between the cortex and the apparent origin of the nerves at the base of the brain or from the latter point to their peripheral terminations. The first class may be subdivided into *cerebral* (between the cortex and the nuclei), *nuclear,* and *fascicular.* The second class may be subdivided into *basal* and *orbital.*

Cerebral Lesions.—These can produce only conjugate or associated deviations, because this region presides over the yoked action of the ocular muscles only. Stimulation of the right hemisphere causes deviation of the eyes to the left, and stimulation of the left hemisphere causes deviation of the eyes to the right. A lesion which would act destructively to this region, either directly or distantly, would paralyze this movement, and the eyes would be turned in the opposite direction by the action of the opposing conjugate acting muscles. The eyes would therefore be turned toward the lesion and away from the paralyzed side of the body. Irritating lesions

would have the opposite effect, and here the eyes would be turned away from the lesion and toward the convulsed side of the body.

NUCLEAR PALSIES.—Nuclear lesions may give rise to "conjugate lateral paralysis" (Swanzy) the relation of the deviation to the site of the lesion being the reverse of that arising from a cerebral lesion, as the sixth nerve presides over the conjugate action of the external rectus muscle of the same side and the internal rectus of the opposite side. Convergence would be retained, but the individual action of the externus would likely be paralyzed.

Paralysis of convergence from a lesion of the convergence centre, which is supposed to be in the aqueduct of Sylvius, is occasionally seen. It may be associated with paralysis of accommodation, and is at times accompanied

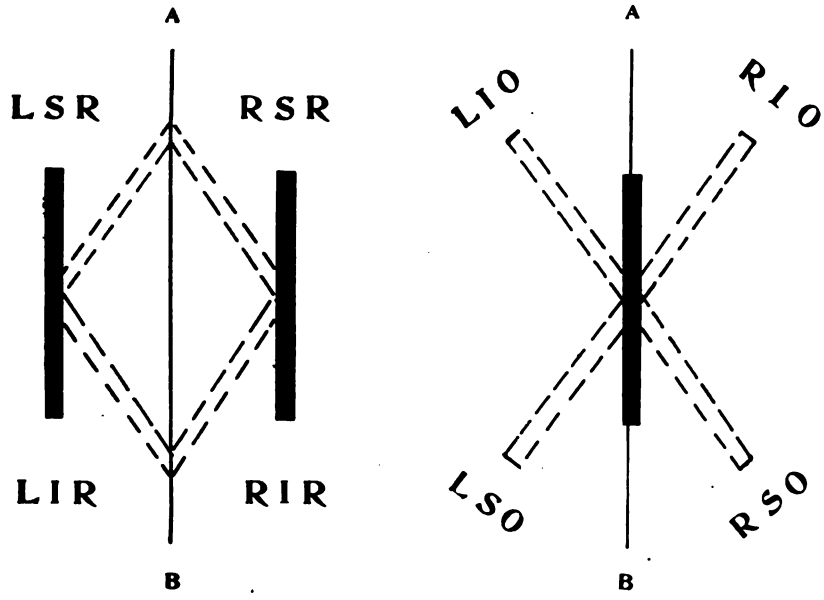


Fig. 374.—Mnemonic scheme of ocular paralyses. (WERNER.)

by paralysis of sursumvergence and deorsumvergence. It is most frequently met with in disseminated sclerosis.

NUCLEAR PALSY, OR OPHTHALMOPLEGIA, is the result of a lesion affecting the nuclei of the ocular muscles in the floor of the fourth ventricle and in the aqueduct of Sylvius. The external muscles alone may be involved (external ophthalmoplegia) or the internal muscles alone (internal ophthalmoplegia), or both groups together may be affected (mixed ophthalmoplegia). It may be congenital or acquired. The acquired type appears in two forms, acute and chronic.

Acute Ophthalmoplegia is a rare disease, usually beginning with headache, vomiting, and dizziness, followed by rapid paralysis of all the ocular muscles. The disease ends speedily in death. The lesion is due to hemor-

rhagic extravasations. The cause is usually alcoholism or, more rarely, other poisons and traumatism.

Chronic Ophthalmoplegia may be due to syphilitic arteritis or may occur as a complication of tabes, disseminated sclerosis, general paralysis, multiple neuritis, and exophthalmic goitre. It also occurs after diphtheria and, rarely, after measles, scarlet fever, and influenza; from poisoning by alcohol, tobacco, lead, carbon dioxid, sulphuric acid and ptomains; and as the result of traumatism. The affection begins by involvement of one or more of the external muscles and progressively involves all. The internal muscles are usually spared, but may be affected independently or in conjunction with the external muscles. The third nerve is most frequently affected, but the degree and frequency with which the levator is involved

Fig. 375.—Ophthalmoplegia. (AUTHOR.)

The patient is attempting to open his eyes.

is moderate. It is usually bilateral. The prognosis, as is to be inferred from the name, is unfavorable. Treatment must be directed to the cause. The indications will generally best be met by the use of large doses of potassium iodid and of strychnin.

FASCICULAR PALSIES.—Ocular paralyses due to lesions situated between the nuclei and the base of the brain are frequently associated with paralysis of the opposite side of the body, the so-called alternate paralysis. The differential diagnosis between this group and those due to lesions of the nuclei and of the base is often impossible. Cases with upward deviation of one eye and downward deviation of the other have been seen but rarely, with lesions in the crura cerebelli.

PERIPHERAL PARALYSIS.—The most common causes of peripheral paralysis are syphilis and rheumatism. Whether the paralyses met with

in diabetes, diphtheria, herpes zoster ophthalmicus, influenza, and the various forms of poisoning are not at times peripheral, and not always nuclear, is still undecided.

Basal Lesions.—These are the most frequent causes of ocular palsies, and are usually of such a nature as to operate through direct pressure, as in meningitic exudate or tubercle, neoplasm and gumma either of the meninges or of the brain-substance, ostitis and periostitis, hemorrhage, aneurism, or fracture.

Any or even all of the muscles of one or of both eyes may be affected and other cerebral nerves may be involved. When ocular palsies are accompanied by monocular blindness; bitemporal hemianopsia or homonymous hemianopsia, associated with the hemianoptic pupillary reaction sign; or

Fig. 376.—Ophthalmoplegia. (AUTHOR.)

The lids are held open to show the position of the eyeballs.

where, in the oculomotor palsies, the internal muscles are not involved, they are usually of basal origin.

Recurrent Third-Nerve Paralysis.—This is a rare affection usually seen in youth and in the female sex. It is always monocular, and in the recurrences the same nerve is always affected. The attacks last from days to months, and the intervals vary from a period of from ten days to a year's time. In the few autopsies recorded the lesion has been of the nerve-trunk.

A similar affection of the sixth nerve has been observed.

Orbital Lesions affect the muscles and nerves principally mechanically. The lesion may be aneurism, neoplasm, or inflammation of the orbital tissues or of the accessory sinuses, periostitis, fracture at the sphenoidal foramen, and foreign body in the orbit.

Prognosis in Ocular Paralyses.—This, of course, varies with the cause. It is most favorable in the peripheral forms. The tendency to relapse must be remembered in connection with those of spinal origin. The duration of the paralysis is nearly always lengthy, and when treatment fails a secondary contracture of the opposing muscle gives rise to a squint, which has the effect of equalizing the angle of deviation for all directions in which eye is turned, thus practically converting the squint into a comitant one.

Paralysis of the Seventh Nerve (Facial, or Bell's, Palsy).—When the lesion producing this affection is nuclear, supranuclear, or infranuclear, the upper distribution of the facial nerve (orbicularis palpebrarum and frontalis muscle) is involved, this involvement is greater and more persistent when the lesion is nuclear, or intranuclear, and in these conditions may cause lagophthalmos—an inability to close the lids, which even in sleep remain open (Spiller). When associated with paralysis of the trigeminus there is danger of ulceration of the cornea supervening, as this membrane is then deprived both of its trophic innervation and of its external protection, and is in consequence easily injured by the foreign matter which readily finds lodgment there.

Treatment is of the cause, the indications being met in a large percentage of cases by the administration of the iodid of potash in ascending doses and of mercury used in inunctions, hypodermically, or internally. In the later stages strychnin in heroic doses may do good. Electricity, either in the form of the constant or the faradic current, is of value. A weak constant current of from 1 to 3 milliampères will be found most serviceable. The negative pole should be applied to the closed lid and the positive one to the temple. Mechanical exercise has proven of service. It consists in seizing the anesthetized conjunctiva over the insertion of the paralyzed muscle and rolling the eye as far as is possible in the direction of the action of the muscle. This is to be repeated several times. When, in facial and trigeminal palsy, impairment of the cornea is threatened, the operation of tarsorrhaphy is indicated.

Paralytic Mydriasis occurs as the result of lesions affecting the nucleus of the third nerve or of the nerve itself. Mydriasis also occurs as the result of interruption in the transmission of light-impulses from the retina, as, for example, in optic-nerve atrophy; but this is not a true paralytic mydriasis. The pupil is partly dilated and is susceptible of further dilation by the instillation of a mydriatic.

The conditions giving rise to paralytic mydriasis are cerebral disease (especially general paralysis of the insane), lesions at the base of the brain and those involving the nucleus, thrombosis of the cavernous sinus, tabes dorsalis (as the result of third-nerve palsy), orbital disease producing pressure on the ciliary nerves, increased intra-ocular pressure (intra-ocular growths and glaucoma), blow received upon the globe, diphtheritic and ptomain poisoning, apoplectic coma, syphilis, and mydriatics when used internally or locally. When paralysis of the iris is associated with paraly-

sis of the ciliary muscle, the condition is known as *ophthalmoplegia interna.*

Paralytic Miosis is most frequently met with as a symptom of tabes dorsalis and is due to disease of the ciliospinal centre. Later in the course of this affection the so-called Argyll Robertson pupil is frequently present as a result of the upward extension of the disease process. It is also caused by injury to the cervical sympathetic or as the result of pressure upon the nerve from aneurism or from enlarged lymphatic glands.

Paralysis of Accommodation (Cycloplegia).—Paralysis of the ciliary muscle is usually associated with involvement of one or more branches of the third nerve. Absolute palsy, aside from that induced by cycloplegics, is most commonly due to diphtheria. It usually comes on during early convalescence, but may be delayed until the sixth week. The symptoms are blurred vision and inability to see to do near work, these symptoms being especially annoying to hypermetropes. Micropsia is occasionally complained of. Paralysis of accommodation is also met with in diabetes, influenza, and mumps (Mandonnet). Paresis or weakness of accommodation is present in all long and exhausting diseases and as the result of excessive venery and masturbation. Unilateral paresis may be caused by syphilis or by disease of the teeth.

The prognosis varies with the cause. Recovery invariably occurs in diphtheritic paralysis. Ascending doses of nux vomica and the use of electricity seem to hasten recovery.

Spastic Strabismus.—Deviation of the eye due to spasm of one or more of the ocular muscles is a rare condition, comprising some cases of intermittent comitant strabismus and of choreic squint. Instances of conjugate deviation the result of irritating cerebral lesions, hysteria, and epilepsy.

Nystagmus is a condition in which there are short, rapid, rhythmic, involuntary movements of the globe. These movements may be horizontal, vertical, or rotary, and are nearly always bilateral. Nystagmus is either congenital or acquired. The former type is usually associated with defective vision due to optic-nerve degeneration, opacities in the media, patches of chorioiditis, and albinism.

Acquired nystagmus may be symptomatic or occupational. The former is frequently a symptom of disseminated sclerosis and of Friedreich's ataxia, and may be due to lesions in the cerebrum or cerebellum.

Occupational nystagmus is met with in miners engaged in "undercutting," requiring the continuous upward turning of the eyes for long periods of time (Snell). The condition has been met with in various other occupations and is to be looked upon as being due to fatigue of the muscles and exhaustion of their innervation (Baer, Snell).

TREATMENT is of avail only in the occupational type, and consists of abstinence from work for a time, with absolute rest to the eyes and correction of existing refraction and muscular errors, or in a complete change of occupation.

Deviation of the Visual Axes of Non-paralytic Origin (Heterotropia).—To this class of muscular anomalies the term *comitant* strabismus[1] is given, because of the distinguishing feature that the angle of deviation remains the same whether the eyeballs are turned in the direction of the squint or in the opposite direction; or, in other words, that the affected eye follows its fellow fully in all its movements.

The terms *convergent* (in), *divergent* (out), *sursumvergent* (up), and *deorsumvergent* (down) strabismus are used to distinguish them according to the direction assumed by the deviating eye.

The following nomenclature has been given by Stevens to the condition of equilibrium and to the various deviations therefrom:—

Orthophoria, the condition in which muscular equilibrium is maintained with the minimum of nervous effort.

Heterophoria, a tending of the visual lines from parallelism.

Heterotropia, a deviation of the visual lines from parallelism in such a manner that they cannot habitually be united at the same point of fixation.

The class of heterophorias includes:—

Esophoria, a tending of the visual lines inward.

Exophoria, a tending of the visual lines outward.

Hyperphoria, a tending of the visual lines of one eye in a direction above its fellow. (The term is a relative one and must be prefixed by the designating word *right* or *left*.)

Intermediate tendencies are termed *hyperesophoria* (right or left) and *hyperexophoria* (right or left).

Esotropia, a deviation of the visual lines inward.

Exotropia, a deviation of the visual lines outward.

Hypertropia, a deviation of one visual line above the other. (The term is a relative one and must be prefixed by the designating word *right* or *left*.)

Intermediate deviations are termed *hyperesotropia* (right or left) and *hyperexotropia* (right or left).

Variations of the equilibrium, which may or may not be consistent with parallelism of the visual lines, but in which, with the least innervation of the eye-muscles, the visual lines would tend below or above the most favorable plane for the minimum effort, are termed, respectively, *cataphoria* and *anaphoria*.

A rotary tendency, due to insufficiency of the oblique muscles, has been described by Savage and has received the name *cyclophoria*.

General Symptoms.—Aside from the above characteristic symptom, which alone would serve to distinguish this form of squint from the paralytic variety, there is usually absence of diplopia, equality of the primary and secondary deviations, and amblyopia of the deviating eye by which to differentiate them.

Cause of Heterotropia.—Deviations of the line of vision may have their origin in anomalies of refraction, in the structure and insertion of the ocular muscles, and in the development of the orbits.

---

[1] The terms "strabismus" and "squint" have been retained because of their general employment, but the terms introduced by Stevens are to be preferred.

Comitant Convergent Squint (Esotropia).—There are three varieties recognized: *permanent monocular, permanent alternating,* and *periodic.*

Permanent monocular squint is the most common. It usually makes its appearance at the age at which the child first begins to be interested in near objects, such as toys, picture-books, etc.—about two years of age. It may, at first, be periodic, but soon becomes permanent and monocular. Its origin is almost invariably attributed by the laity to whooping-cough, fright, imitation, or to the attraction of the child's sight to some object hanging overhead. The most frequent, but by no means the only cause, is hypermetropic refraction. This operates to produce it through the desire for distinct single vision, which can be attained only by excessive convergence of the visual axes, with its associated accommodative act. Normally, for each angle of convergence an equal degree of accommodation is employed. In the hypermetrope this balance is disturbed from the beginning, since, in order to see distinctly at a distance, the hypermetrope must accommodate to the degree of his hypermetropia and still maintain parallelism of the visual axes. Now, when it comes to viewing an object at near range, say forty centimetres, one of two conditions must prevail: either he must be satisfied with blurred, but single, vision (both eyes converged to the object, but accommodation insufficient), or he may secure distinct vision (both eyes accommodated for the object, but convergence excessive), which will produce double vision unless he permits one eye to turn in, and this is the solution of the difficulty which the patient instinctively accepts. Other causes of convergent squint are:—

1. Congenital weakness of the external rectus muscles, which would be particularly apt to produce convergent squint if from any cause one eye is amblyopic, or the retinal image is distorted or faint as the result of opacities in the cornea or other of the refractive media.

2. A high positive quality of the angle gamma would tend to the production of a convergent squint.

3. Failure of development of the fusion centre (Worth).

In convergent squint the result of hypermetropia, the deviating eye, in more than half the cases, is found to be amblyopic. The causal relationship between the squint and the amblyopia still remains in dispute, some authorities looking upon the amblyopia as the result of the squint from disuse of the eye, and others regarding it as the cause of the squint by removing the stimulus to convergence which would be excited if there was a tendency to diplopia. The absence of diplopia in comitant strabismus may be due to the suppression of the retinal image, or it may arise from the development of new identical points in the retina of the deviating eye.

Comitant Divergent Strabismus (Exotropia).—The same varieties of this form of squint may be recognized as of convergent strabismus. The permanent monocular variety is the most common, but the alternating is more frequent than in convergent squint. As a rule, divergent squint appears later in life than does convergent, because it is usually associated

with high degrees of myopia, which condition, for the most part, is an acquired anomaly. It is also seen as a sequel to insufficiency of convergence—a condition which may be met with in any state of refraction. Excessive divergence of the axes of the orbit in the course of the development of the skull may lead to divergent strabismus (Weiss). Diplopia is of more frequent occurrence than in convergent squint, probably because binocular vision has been maintained to a later period of life. The *angle of squint* is the angle which the visual axis of the deviating eye forms with the direction which it should have in a normal condition (Landolt).

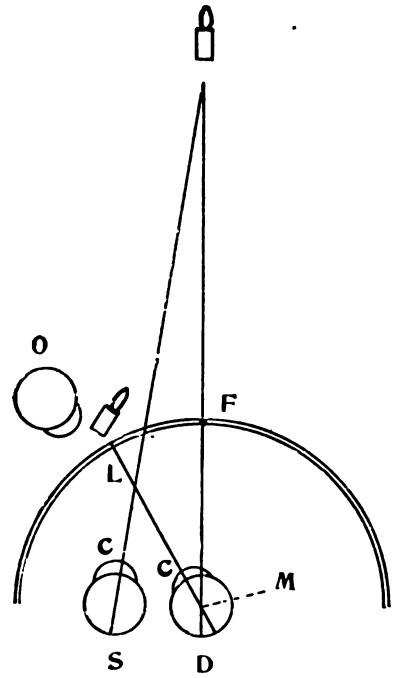


Fig. 377.—Measurement of the angle of squint by the perimeter.

D, Squinting eye. *S*, Fixing eye. *O*, Observer's eye. *L-M-F*, Angle of squint. *C, C*, Centre of corneas.

Determination of the Angle of Squint.—Much ingenuity has been displayed in devising methods for the ready estimation of the angle of squint, and of these methods three will be detailed—those by the perimeter, by prisms, and by the linear method, or strabismometry.

By the Perimeter.—The patient is seated before the perimeter, with both eyes uncovered, and is directed to look at the button on the perimeter or at a candleflame held on the visual line at a greater distance. While so doing a second lighted candle is carried along the arc of the perimeter, and the examiner, with his eye placed directly behind this candleflame, notes when the image of the flame falls upon the centre of the cornea of the deviating eye, and when this has come to pass the angle of the squint may be read

off in degrees. The centre of the cornea is employed because it is impossible to determine the visual axis by this method; consequently the result obtained is only approximately correct, as it does not take into consideration the difference which almost invariably exists between the optic axis and the visual axis and the resulting angle formed by the crossing of these two lines at the principal optical centre. In order to be exact, this angle must also be estimated and its value added in convergent squint and deducted in divergent squint from the angular determination.

By Prisms.—This method is applicable only where diplopia exists spontaneously or can be induced. When diplopia does not exist spontaneously, which is the rule, it may sometimes be induced by placing a red glass before the eye with the better vision; this failing, a prism should be placed vertically before one eye, and, by thus throwing the image on to a different part of the retina, diplopia may often be excited. Prisms are to be held horizontally before either eye of sufficient strength to fuse the two images if diplopia be induced by the first method, or until the images stand one above the other, if diplopia be induced by the second method.

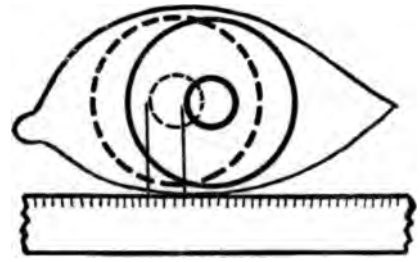


Fig. 378.—Linear measurement of squint.

A second method of employing prisms to measure the degree of deviation is in connection with the cover test. That prism is a measure of the squint which maintains the visual axes directed to the fixing object held about forty centimetres before the eye when the cover is transferred from one eye to the other.

Linear Method.—A ruler is held close against the lower lid of the squinting eye, and the divisions corresponding to the centre of the cornea when the eye is made to fix a distant object, and when it again is allowed to assume its abnormal position, are noted. The difference is a measure of the squint. A special instrument called a strabismometer may be used. This test is not without value in determining the length of the piece of muscle desirable to remove in performing advancement with resection of the muscle in high degrees of squint, but, as is insisted upon by Landolt, it is manifestly incorrect to speak in linear terms of an angular quantity.

TREATMENT OF STRABISMUS.—The vast majority of cases of strabismus having their origin in an error of refraction, the first step in their treatment will consist in the correction of that error. The total anomaly should

be determined under complete paralysis of the ciliary muscle, secured preferably by the use of atropin, and the glass which is found to correct it should be worn constantly. By the aid of ophthalmoscopy and retinoscopy the refraction *status* can be quite accurately determined, even in very young children, and at the age of three and a half years they may be given glasses to wear. Experience has demonstrated that, by this method of treatment, the visual axes are rendered parallel in cases of moderate degree. The parallelism is maintained so long as the glasses are worn, the eye reverting to its faulty position as soon as they are removed. In cases where convergent squint manifests itself before the age at which it would be deemed advisable to place spectacles on the child, a course of atropinization for the purpose of relaxing the convergence by paralyzing the accommodation should be employed. A weak solution of atropin (gr. j to ℥j) should be instilled into the fixing eye twice daily for a period of several months, care being taken not to get the toxic effect of the drug and to shade the eyes from bright light. In a few cases the primary effect of the atropin will be found to increase the squint as the result of the excessive accommodation innervation which is induced by the attempts of the patient to see distinctly (Savage).

Orthoptic Treatment.—For success with this method diplopia must exist; and, if not present spontaneously, it must be induced. This condition obtaining, a stereoscope, in which the prisms have been replaced by convex spheric lenses of 6 D. strength, and in which the distance between the stereoscopic pictures for the two eyes may be increased or lessened at pleasure, is employed to stimulate binocular vision. The test pictures must be placed at the focus of the lenses so that they will be plainly seen without accommodation, and, associated convergence being stimulated,—if the visual axes be parallel, and the pictures be not too far apart,—the two images will be fused into one having the third dimension. Where the visual axes converge, the pictures must be approximated until both eyes see simultaneously. After this has been accomplished actual fusion and restoration of binocular vision may be expected. Where there is unequal acuity of vision in the two eyes, difficulty will be experienced in inducing the eye with the reduced visual acuity to perceive the picture corresponding to that eye. Equalizing the visual acuity of the two eyes by fogging the sight of the better-seeing eye, by using a higher plus lens than is before the other eye, or repeatedly and momentarily excluding the better eye, will be found of assistance. The amblyoscope of Worth also will prove useful in developing the vision of amblyopic eyes and in bringing about binocular vision if employed during the years the fusion faculty is developing—that is, before the child is six years of age. The patience and time required to secure results, and the limited field of usefulness of this form of treatment, have interfered with its securing the position it deserves. It will also be found of service in restoring binocular vision where operation has produced spontaneous diplopia.

Operative Correction of Strabismus.—Should the above methods fail to restore parallelism to the visual axes, the question of operative intervention must be considered. No unanimity exists among surgeons, either as to the method to be pursued or as to the age at which it should be undertaken. Without entering into a discussion of the reasons it may be stated that the operation of advancement of the tendon of the muscle more nearly restores the normal physiologic and anatomic relations than does tenotomy. However, owing to the ease with which it may be performed, tenotomy is the prevailing method of procedure. No operation should be performed until the patient has arrived at an age at which a comprehensive study of the conditions can be made and the co-operation of the patient can be obtained in restoring binocular vision. Nor should it be delayed until time has effaced the memory of binocular vision. As to the results to be expected from the performance of tenotomy, it may be stated for general guidance that a tenotomy of a single internal rectus muscle, without a free dissection of the reflected fibres of the capsule of Tenon, will correct about 15 to 20

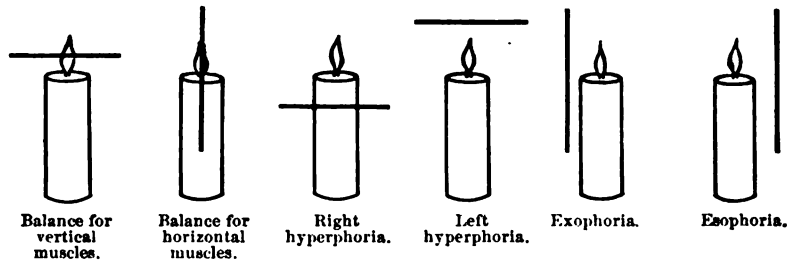


Fig. 379.—Relative positions of the line of light and the candle-flame in the Maddox rod test.

The rod is before the right eye.

degrees of deviation; and a double tenotomy, from 20 to 35 degrees. The effect will be somewhat less after the same operation on the external muscles. For higher defects a tenotomy with advancement of one or both of the opposing muscles may be required.

Latent Deviation (Heterophoria).—So great is the antipathy to double vision that a weakness of a muscle of sufficient degree to become at once manifest, when the incentive to binocular vision has been removed, will be continuously overcome, but at the expense of exhausting nervous innervation.

The muscular equilibrium may be disturbed by either an actual or a relative weakness of one or more muscles, such weakness causing a tendency toward deviation of the visual axes.

For the determination of these tendencies it is necessary to remove the incentive to binocular vision. Various methods for accomplishing this have been devised, but only the more useful will be considered.

Maddox Rod Test.—A cylinder of glass, or some modification thereof, which has the effect of converting the image of a point of light into a

line of light having a direction at right angles to the long axes of the cylinder is placed before the eye, with its length horizontal, if it be desired to test the lateral muscles, and vertical if the vertical muscles are to be tested. A small light is placed at a distance of five metres in front of the patient. The line of light replaces, in the eye before which the rod is placed, the image of the flame. If the visual axes be parallel, the line of light will bisect the flame. If exophoria exist, the line of light will lie to the opposite side of the flame to that of the eye before which the rod has been placed (crossed diplopia). In esophoria it will lie on the side corresponding to the eye before which the rod has been placed (homonymous diplopia). In hyperphoria it will lie above or below the light, and will be designated right or left according to whether the image of the right or left eye be the lower.

Equilibrium Test (von Graefe's Test).—A prism of 8° or 10° is placed with the base down before either eye and a small light at twenty feet (six metres) distance is used as the test-object. The prism removes the tendency to fusion by displacing the image belonging to the eye before which the prism has been placed to a point below the fovea, in consequence of which it will be projected above the image belonging to the other eye. If orthophoria for the lateral muscles exists, the images of the two eyes will stand one directly above the other. If exophoria be present, the displaced image will lie to the opposite side of the image belonging to the other eye from that before which the prism has been placed. If esophoria be present, it will lie on the corresponding side.

To prevent fusion of the images in testing the balance of the vertical muscles, a somewhat stronger prism should be employed with the base of the prism held toward the nose. If hyperphoria exists, one light will appear higher than the other. Hyperphoria of that eye exists whose image is the lower. In all these tests the degrees of the prism required to bring the lights into alignment is a measure of the deviation. The equilibrium test is now usually made with the phorometer of Stevens, which consists of a pair of 5° prisms so mounted, in rotary cells, one before each eye, that their bases are opposed when the prisms are rotated to the vertical position and are in apposition when in the horizontal position. A scale is so affixed to the circumference of each cell that the amount of heterophoria, which is determined by the amount of rotation requisite to bring the lights into alignment, may be at once read off. Accessory cells in which are mounted a rotary prism and a Maddox rod and which contain grooves for trial lenses complete an instrument which is suited for testing the refraction and muscular anomalies.

Parallax Test (Duane).—While fixing a small light placed at a distance of twenty feet (six metres), and as close to the plane of the wall as is possible, one eye is momentarily covered by a card, after which it is rapidly transferred to the other eye. If heterophoria exists, an apparent movement of the light will take place. If this movement takes place in the direction of the eye to which the card is transferred, exophoria is present; if in the

opposite direction, esophoria exists. If the movement is up, there is hyperphoria, designated by the side to which the card has been transferred; if down, there is hyperphoria, designated by the side from which the card has been transferred. The degree of the prism which neutralizes the apparent movement is a measure of the deviation.

COBALT-GLASS TEST.—This simple test will be found to uncover even very low degrees of heterophoria. A cobalt glass or the superposed red and the blue glass usually found in trial cases is placed before one eye, and in front of this is held a card. After waiting twenty or thirty seconds the card is slowly withdrawn laterally from in front of the pupillary space; and when the edge of the card has passed the visual axis two lights—one of a cobalt color, the other of the color of the flame—will be observed, if heterophoria exist. If orthophoria is present, but one light of the color resulting from the superposition of the two images will be seen. That prism or combination of prisms with which but one light is observed is a measure of the degree of heterophoria.

In all of the above tests the test-object should by preference be a luminous disc from one-half to one centimetre in diameter.

The above tests serve to indicate the tendency of the visual axes when the accommodation is at rest. To determine this tendency when it is being used—that is, at reading distance—either of the first two tests may be employed, using for the test-object a three-millimetre dot on a white card, and a 30 D. convex cylindric lens in place of a Maddox rod.

COVER TEST.—One eye is covered by the hand of the examiner while the other fixes a pencil-point held in front of the face at a distance of forty centimetres. After a few moments the hand is transferred to the other eye, when, if heterophoria exists, the uncovered eye will be seen to make a movement in order to fix the pencil-point. If the eye moves outward, it must have been deviated inward while under cover, indicating insufficiency of the external rectus, or esophoria. If it moves inward, it must have been deviated outward while under cover, indicating exophoria. If it moves either upward or downward, it indicates hyperphoria. If no movement takes place, the visual axis must have maintained the same direction as that of the other eye, indicating orthophoria.

TEST FOR CYCLOPHORIA (SAVAGE).—A Maddox double prism is placed before one eye with the basal line bisecting the pupillary space, horizontally, the other eye being covered. The patient is directed to look at a horizontal line drawn on a card, held at forty centimetres. The effect of the prism is to produce monocular diplopia, two parallel horizontal lines appearing. The other eye is now uncovered, when a third line, placed between the other two, will come into view. The naked eye is the eye under test. If there is harmonious action of the oblique muscles, the three lines will appear parallel. If there is want of harmony, it will be shown by obliquity of the middle line. With the prism before the right eye, if the right end of this line dips, there is insufficiency of the left superior oblique; if the left end

dips, there is insufficiency of the left inferior oblique. With the prism before the left eye, if the left end dips, there is insufficiency of the right superior oblique; if the right end of the line dips, there is insufficiency of the right inferior oblique.

Having determined the direction assumed by the visual axes when deprived of the extra innervation called forth by the desire for single vision, it remains to determine whether this abnormal tendency is associated with deficient or excessive adducting or convergence power; deficient or excessive abducting or divergence power; or deficient or excessive supra- or infraducting power.

The *power of convergence* can be measured in metre angles (see page 123) or by determining the near point of convergence, and also by the power of the internal recti muscles to overcome the diplopia produced at twenty feet (six metres) by prisms placed with the bases out before the eyes. In order to estimate the *near point of convergence* it is only necessary to approach a pointed lead-pencil to the eyes along the median line and measure the distance from the eyes at which the point begins to appear

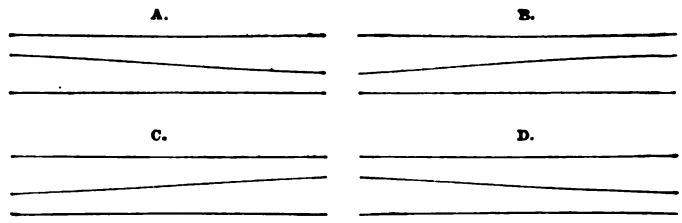


Fig. 380.

A, Insufficiency of the left superior oblique. *B*, Insufficiency of the right superior oblique. *C*, Insufficiency of the left inferior oblique. *D*, Insufficiency of the right inferior oblique.

double. *Prism convergence* varies greatly within normal limits, ranging from 25 to 90 degrees or even more.

The *power of divergence* can be measured only by the power of the external recti muscles to overcome or fuse the diplopia produced by prisms held with the bases in before the eyes. This normally equals from 7 to 8 degrees.

It will be found that the adducting power determined at the first trial is frequently very far below the above-mentioned limits even in those with whom a few trials develop a normal degree or who possess the full number of metre angles of convergence, while the abducting power is a fairly constant quantity in repeated tests. Supraduction is measured by the degree prism which, placed with its base down before either eye, can be overcome; likewise infraduction is measured by the degree prism which can be overcome when placed with its base up before either eye. Both should equal 2 to 3 degrees.

The following is, in abstract, Duane's finding of associated conditions in excessive and insufficient convergence and divergence power:—

In *Insufficiency of Convergence* there is either orthophoria or a tendency to exophoria at twenty feet (six metres) and marked exophoria at near distances. The near point of convergence may be seven to ten centimetres. The adducting power is low and difficult to develop, and the abducting power is normal, slightly increased, or decreased.

In *Excessive Divergence* there is exophoria for near and far distances. The convergence near point is nearly normal. The adducting power is low or may be normal and the abducting power is excessive.

In *Insufficiency of Divergence* there is esophoria for distance and orthophoria or possible exophoria for near. The convergence near point is normal. The adducting power is normal and the abducting power is greatly diminished.

In *Excessive Convergence* there is orthophoria or slight esophoria for distance and marked esophoria for near. The near point of convergence is very close to the eyes. The adducting power is high and the abducting power may be low. Disturbance of vertical equilibrium affects the lateral equilibrium and may aggravate or even originate lateral deviations.

SYMPTOMATOLOGY.—Disturbance of the ocular muscular equilibrium gives rise to a great variety of symptoms, ranging from ocular pain with headache to a symptom-complex simulating grave organic nervous disease. The more usual group of symptoms is headache, basal or extending from the eyes to the occiput; pain between the shoulder-blades; confused vision frequently called forth by car-riding, sight-seeing, and shopping, and often causing anxiety in the turmoil of crowded thoroughfares; consciousness on the part of the patient that the ocular muscles are not working in harmony (in some instances the patient will state that the sensation is as though he had lost control of the eyes and that they had momentarily turned either in or out); nausea and dizziness and even vomiting if the strain is continued. Restlessness is frequently marked, especially in children. There can be no doubt that neurasthenia does result from uncorrected muscular anomalies, and it is conceivable that epilepsy, migraine, and chorea might be induced in the extremely neurotic with strong predisposition to these affections. The local symptoms do not differ greatly from those excited by anomalies of the refractive media, namely: blepharitis, clonic blepharospasm, hordeola, and congested conjunctiva, at times more pronounced over the insertion of the faulty muscle.

TREATMENT.—The first measure to be adopted, in the treatment of these troublesome disturbances, is fully to correct any error of refraction that may exist, the only exceptions to this rule being those cases of insufficiency of convergence associated with hypermetropia, where it is advisable to stimulate convergence through the accommodation, which may in a measure be accomplished by correcting only so much of the hypermetropia as is consistent with the age of the patient and the work required of the eyes; and in those cases of myopia associated with divergence insufficiency, where convergence should be lessened by diminishing the accommodation, which may be accomplished by partially correcting the myopia.

In connection with the local treatment general treatment is always demanded. Many of these sufferers are the offspring of neurotic parents or come of phthisical stock. The use of nux vomica for insufficiency of convergence and of hyoscyamus for insufficiency of divergence will be found to be of only temporary benefit.

The symptoms excited by low degrees of heterophoria are often allayed by the wearing of prisms. This is particularly true of vertical tendencies, and, in cases where this is of moderate degree, about two-thirds of the whole amount may be corrected with the expectation of thus unmasking some of the latent error. When this occurs it should again be corrected, and this procedure can be repeated until the error is a constant one, when an operation can, with understanding, be performed.

In *weak adduction* it is well not to correct the total amount of exophoria, as it is apt still further to increase the convergence insufficiency. Usually the prisms need be worn only at near work, and for this purpose they may be put into fronts, a supplementary frame to be hooked on the front of the spectacles containing the refraction correction. Esophoria may be totally corrected in *weak abduction*. There are optical and physical limits to the degree possible to be corrected by prisms, and, as a rule, 3.5° prisms before each eye is about all that can be worn, and these are apt to be uncomfortable, owing to their weight, to the chromatic aberration, and to the reflection from their surfaces.

Several methods have been proposed for *strengthening adduction* and *abduction*. The plan most in vogue is to practice fusing of images produced by placing prisms before the eye in such a manner that the false image is displaced in the direction of action of the muscles to be strengthened. A small flame having been placed at a distance of six metres, the patient is directed to begin with the weakest prism which produces diplopia and endeavor to fuse the images. When this has been accomplished the eyes are to be momentarily closed, and, as soon as on opening the eyes fusion occurs without conscious effort, the strength of the prism is to be increased. Exercise of convergence or adduction requires the use of prisms placed with their bases toward the temple. Care must be taken to have the prism exactly horizontal, which may be told by the lights being on a horizontal line, or fusion will be difficult or even impossible. Such exercise should not be continued longer than ten minutes at a time nor repeated oftener than twice a day. When adduction has reached about 50 degrees no further increase is necessary, but prisms of this strength should be practiced with thereafter. In order to exercise divergence or abduction, the weakest prism which produces diplopia at six metres should be placed before the eye. If fusion is impossible the light should be approached, and, when the images merge, the distance from the light should be increased. A divergence power of 8 degrees should be striven for. Adduction may also be strengthened by having the patient view the tip of a pencil as it is approached to the eyes. As soon as it appears double he should look momentarily at a distant light;

this is to be repeated for about ten minutes, twice a day. Should all such means fail to correct the deviation or to relieve the subjective symptoms, operative intervention is called for.

Savage seeks to *exercise* the *weak obliques* by the use of cylindric lenses placed at oblique axes before the eyes. Either convex or concave lenses may be employed. If convex, the axes must be placed in the lower temporal quadrant for insufficiency of the superior obliques, and in the lower nasal quadrants for insufficiency of the inferior obliques. The effect is increased by revolving them from the vertical, the maximum effect being secured at 45 degrees therefrom. The exercise should be commenced with 0.50 D. lens placed before both eyes, and increased each day a 0.50 D. up to 3 D. The patient is directed to fix a distant light for three minutes, at the end of which time the cylinders are revolved symmetrically 15 degrees and the light is again viewed. The exercise may be repeated twice a day.

In *convergence weakness* the proper operation is *advancement* of the tendon of the internal rectus muscle or advancement of the capsule of Tenon. The latter operation will be found useful where the exophoria does not exceed 8 degrees for near.

In *convergence excess* a carefully performed tenotomy of the internal rectus is called for.

In *divergence weakness, advancement* of the tendon of the external rectus or of the capsule of Tenon is the proper procedure.

In *divergence excess* a carefully performed *tenotomy* of the external rectus will be found adequate.

OPERATIONS ON THE OCULAR MUSCLES.

Tenotomy.—General anesthesia is required only in patients under eight years of age. For local anesthesia a 4-per-cent. solution of cocain will be found efficient, a few drops being instilled into the conjunctival sac before the operation and into the subconjunctival tissues through the conjunctival incision from time to time during the operation. A stop speculum having been inserted, the conjunctiva is incised with a pair of tenotomy scissors a little to the corneal side of the insertion of the tendon. The underlying fibres of the capsule of Tenon are then snipped sufficiently to expose the insertion of the tendon and so permit of the strabismus-hook being inserted beneath the tendon. The tendon is then severed by a series of cuts made with the scissors between the hook and the globe. To make sure that no strands have been left undivided the hook should be reinserted and, with its point kept in contact with the sclera, it should be swept above and below the limits of the attachment. If a thorough division has been made, the hook will appear subconjunctivally at the margin of the cornea. A conjunctival suture is unnecessary. Panas advises stretching of the muscle before severing it. The eye should be lightly bandaged for about forty-eight hours. Immediately after the operation the excursions of the eyeball should be full, and, if the internal rectus has been cut, convergence to five

centimetres should be possible; if it is not, then the muscle should be brought forward by a suture, one end of which passes through the tendon, the other through the conjunctiva close to the cornea. The hemorrhage attending this and other operations made on the ocular muscles will be much lessened by the use of a few drops of adrenalin solution (1 to 3000).

ACCIDENTS.—*1. Perforation of the Sclera* during the operation of tenotomy has occurred to many distinguished operators, but such an accident is usually due either to want of skill or to the use of sharp-pointed instead of blunt scissors. The accident is announced by a sinking back of the iris, a loss of vitreous humor, and a reduction in ocular tension. If the accident occurs before the tendon has been severed, it should be cut, provided the wound is anterior to the tendinous attachment, since the contraction of the undivided muscle will cause the wound to gape. If the wound is posterior to the attachment, the muscle should not be cut, since

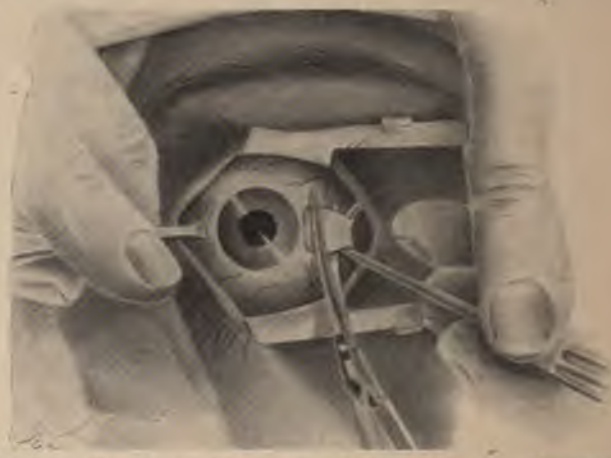


Fig. 381.—Tenotomy of the external rectus muscle. (AUTHOR.)
(Original drawing by DR. R. W. MILLS.)

its normal action will tend to keep the lips of the wound coaptated (Risley). The conjunctiva should be sutured, both eyes should be bandaged, and the patient should be placed in bed. Under favorable circumstances healing will occur without sequelæ. Some of the reported cases have been followed by severe pain, ciliary staphyloma, chorioidal atrophy, reduction in visual acuity, and even panophthalmitis.

2. Excessive Hemorrhage may occur, particularly in hemophiles. It may come from cutting too deeply into the orbital tissues, causing Tenon's capsule to fill with blood and the eye to protrude. Masses of fat sometimes appear in the wound and should be excised. The conjunctiva should be sutured and a pressure bandage should be applied.

3. Post-operative Infection occurs in rare instances, and may lead to tenonitis, panophthalmitis, orbital phlegmon with atrophy of the optic nerve, atrophia bulbi, etc. The condition will require appropriate treat-

ment. A case of diphtheritic infection in the tenotomy wound, occurring in Knapp's practice, ended in panophthalmitis.

4. Unpleasant Sequelæ of tenotomy have been frequently observed. Among them are sinking of the caruncle, exophthalmos, and the conversion of a convergent into a divergent squint.

5. Operation upon the Wrong Eye must be avoided.

Graduated Tenotomy.—The operation of graduated tenotomy has been practiced now for many years, but the question of its value still remains a subject of dispute. The topographic relations existing between the muscles of the eyeball and the eyeball itself; the small result sometimes obtained from a complete division of a tendon; and the slight—at times negative—effect upon muscular anomalies where the graduated operation has been repeatedly performed, all are calculated to inspire distrust in its efficacy.

In those exceptional cases where attention to the error of refraction, to the development of the ducting power of the muscles, and to the health of

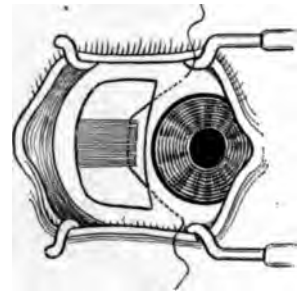


Fig. 382.—Advancement of an ocular muscle, showing the suture in position before tying.

the patient fail to remove undoubted reflex symptoms, it will usually be found that there exists a latent phoria which finally, under prismatic correction of the manifest degree, becomes total and suited to a thorough surgical procedure.

OPERATION.—For this operation special instruments, more delicate in construction than those used for complete tenotomy, have been devised by Stevens. A small fold of conjunctiva, directly over the insertion of the tendon, is seized and snipped transversely so as to make a minute opening. Into this opening the forceps are introduced and a small fold of the tendon, immediately behind its insertion into the sclera, is grasped and snipped. One blade of the scissors is introduced into the opening thus made, and is slipped beneath the tendon, which is then snipped in the direction of one of its borders, to such an extent as may be deemed necessary. The scissors are then turned in the direction of the opposite border, and an equal extent of the tendon is divided. The effect of the operation is then determined, and, if insufficient, more of the tendon is divided. This procedure is repeated until orthophoria is produced.

Advancement.—This operation is severe and tedious, and will be much better done if performed under general anesthesia. A stop speculum is inserted and Tenon's capsule is exposed by a large conjunctival incision, after which it is incised freely over the insertion of the muscle and particularly at the borders, so that the muscle can be lifted from the sclera. One blade of an advancement forceps is then slipped beneath the full width of the tendon and sufficiently far back to permit, if necessary, of excision of a piece of the muscle in advance of its blades, and the forceps closed; if tenotomy of the opposing muscle is called for, it is well to do it at this stage. The tendon is now severed and one needle of a double-armed black

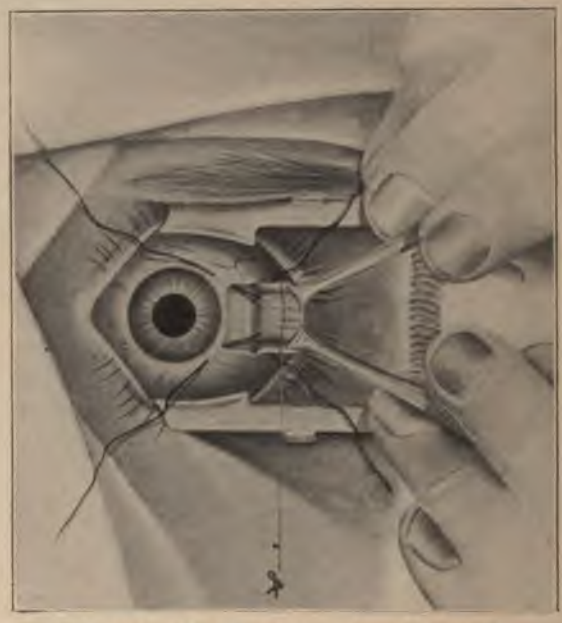


Fig. 383.—Total advancement. (AUTHOR.)

(Original drawing by DR. R. W. MILLS.)

In this operation the muscle, capsule of Tenon, and conjunctiva all are advanced. The tissues external to the dotted line are to be excised.

suture is entered from beneath the tendon and as close to its lower border as is possible. It is then interwoven into the muscle parallel to its cut edge and is brought out as close as is possible to the upper edge of the tendon. The upper needle is then inserted into the episcleral and conjunctival tissues at a distance of about five millimetres above, and the lower needle the same distance below, the horizontal meridian of the cornea, and the ends of the sutures are tied. While tying the sutures the eyeball should be rotated in the direction of the severed muscle. The conjunctival wound should be sutured, redundant tissue having been first excised. The immediate result should be a marked squint of an opposite nature to that for which the operation was done.

Capsular Advancement.—General anesthesia is not necessary. The stop speculum having been introduced, the capsule of Tenon is laid bare by a large conjunctival incision. It is then buttonholed at the lower margin of the tendon at its insertion. A strabismus-hook is inserted into the opening, passed beneath the muscle, and brought out at the upper border of the tendon by incising the overlying capsule. The conjunctiva is then dissected up to the cornea and nearly as far as the vertical meridian. A needle, threaded with a black silk suture, is passed through the episcleral tissue at a point as far back almost as the lower end of the vertical meridian of the cornea, and is brought out into the conjunctival opening, where it is grasped and inserted into the opening previously made into the capsule, under which it is passed as far as the result required warrants, and is there made to emerge through the overlying conjunctiva. After a similar suture has been inserted at the upper margin of the muscle the two ends of each are tied. The immediate effect should be a marked overcorrection of the devia-

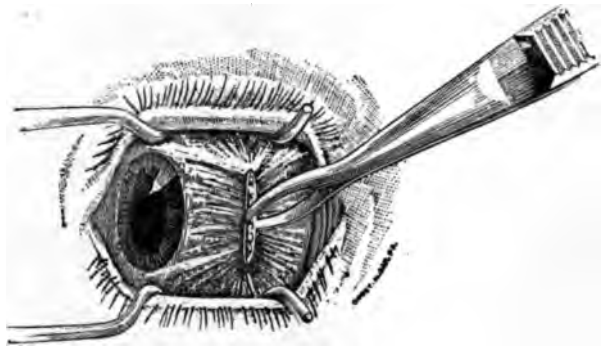


Fig. 384.—Capsular advancement. (Fox.)

tion. In the advancement operations it is well to bandage both eyes and keep the patient abed for forty-eight hours. Ice may be applied for the first twelve hours. The stitches should be removed in five or six days.

[**Fox's Capsular Advancement for Divergent Strabismus.**—This operation, which is done under cocain anesthesia, consists of three steps: (1) tenotomy of both external recti muscles and stretching of the conjunctiva and of Tenon's capsule; (2) the making of an elliptical opening through the conjunctiva and capsule, either on one or both sides; (3) the suturing.

1. Tenotomy of both external recti muscles is performed in the ordinary manner. Tenon's capsule is then stretched by means of traction made upon it with strabismus-hooks. 2. With the retractor forceps (Fig. 384) the conjunctiva is grasped vertically at a point midway between the cornea and caruncle, directly over the internal rectus muscle. That part of the conjunctiva which is grasped by the instrument, together with the subjacent part of Tenon's capsule, is excised. 3. Excision of these tissues leaves a gaping wound. This is to be closed with sutures, which are to be passed through the conjunctiva and capsule of Tenon.—BALL.]

CHAPTER XXI.

ERRORS OF REFRACTION.

By JOHN T. KRALL, M.D., of Philadelphia,

Assistant Surgeon to the Wills Eye Hospital and Dispensary; Ophthalmic Surgeon to the Presbyterian Hospital.

PROPERTIES OF OPTICAL LENSES.

In order more fully to understand the errors of refraction and their treatment we will briefly consider the properties of optical lenses.

Light may be described as that form of radiant energy which may be appreciated by the organ of sight. Not all light-waves are capable of affect-

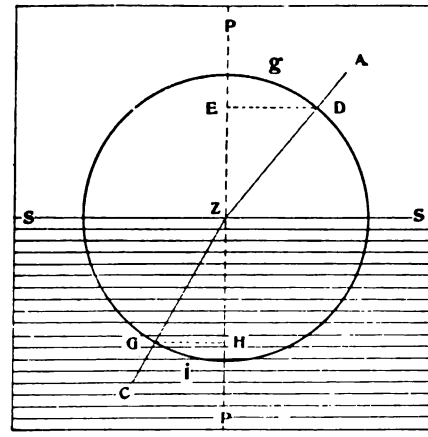


Fig. 385.—Passage of an oblique ray from a rare to a dense medium.

S-S, Plane surface separating the media, the rare medium being above. *A*, Luminous point. *P-P*, Perpendicular. *g*, Angle of incidence. *E-D*, Sine of the angle of incidence. *G-H*, Sine of the angle of refraction. *i*, Angle of refraction.

ing the sight. Light-waves travel through vacuum at the rate of 186,380 miles per second. They do not travel with the same velocity through all substances; the speed is less in water than in air, and is still less in glass. When a ray of light passes from a rarer to a denser medium it will meet increased resistance, which retards its progress. If the transmitting body is less dense than the surrounding media, the vibrations will meet with less resistance; hence they will move with greater velocity. The denser the body, the greater the length of time required to penetrate. The relative length of time it takes light to travel a certain distance in a given sub-

stance is called the *index of refraction* of the substance. If a ray of light enters a new medium in a direction perpendicular to its surface, all parts of the wave-front will be retarded simultaneously and no change in direction will occur. If the wave-front strikes the surface obliquely, one portion still moving in the first medium and retaining its original velocity, while another part of the same wave-front has passed into the second medium, its velocity is impeded, and as a result the wave-front assumes a new direction. This change in direction is called the *refraction of light* (Fig. 385).

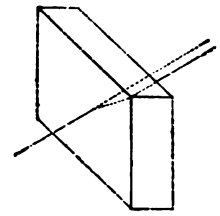


Fig. 386.—Refraction by plane glass.

Rays of light passing from a denser into a rarer medium are deviated from the perpendicular. Should the transmitting medium be rarer than the surrounding medium, rays of light will be turned away from the perpendicular. The deviation depends upon the mediums and the angle of incidence (*g* in Fig. 385). The greater the angle of incidence, the greater the deviation. The deviation diminishes as the angle of incidence diminishes. The index of refraction is obtained by dividing the sine of the angle of incidence (*E-D* in Fig. 385) by the sine of the angle of refraction (*G-H* in Fig. 385). If a plate of glass with parallel surfaces be held in the air, and

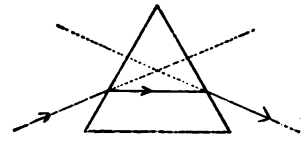


Fig. 387.—Refraction by a prism.

a ray of light be allowed to fall obliquely upon it (Fig. 386), no deviation will occur, as the angle of refraction at the second surface will be equal to the angle of incidence at the first surface; but if the surfaces instead of being parallel were inclined toward one another, as in a prism (Fig. 387), that part of the wave-front that goes through the thicker part of the prism, the base, will be more retarded than the part that goes through the thinner part of the prism, the apex; consequently it would be turned toward the base of the prism. The amount of deviation between the directions of the incident ray and that of the refracted ray is called the *angle of deviation.*

And the amount of angular separation of the two refracting surfaces is known as the *refracting angle.* The amount of deviation produced by a prism varies with the angle at which the incident ray strikes it.

Prisms are numbered by either one of two systems. The *centrad* was introduced by Dennett. It consists in measuring upon a *radian* the amount of linear deflection given to a ray of light which has entered any prism at right angles to its refracting surface, this radian being an arc of a circle which is equal to the radius. A prism which will produce an angular deviation of the one-hundredth part of this arc is called *one centrad.* The *prism-dioptre* was suggested by Prentice. The unit is represented by a linear deflection which is equal to the one-hundredth part of the radius measured on the tangent. A prism situated at one metre's distance from a definite tangent plane and deflecting a ray of light one centimetre along that plane is called a *prism-dioptre.*

If two prisms are placed with their bases together as in *A*, Fig. 388, we then have the fundamental principle of a *converging,* or *convex, lens.* If

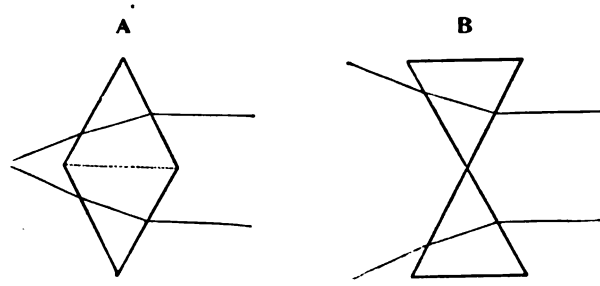


Fig. 388.—Refraction by prisms.

the prisms are reversed and placed apex to apex, as in *B*, Fig. 388, a *dispersing,* or *concave, lens* is formed. A convex lens is simply a series of prisms with bases together; and a concave lens is a series of prisms with their apexes together.

A *lens* may be defined as any transparent medium bounded by surfaces of which at least one is curved. There are six general classes of lenses, as illustrated in Fig. 389.

A *meniscus,* or periscopic lens, is represented in *C* and *F* (Fig. 389).

A line drawn through the centre of curvature of a lens is called its *principal axis.* Rays which do not pass through the optical centre are known as *secondary axes.*

The *optical centre* is a point situated on the principal axis within the lens-substance, except in a meniscus, where it is external. It is found by taking two parallel radii of curvature and connecting the points in which they meet the surfaces. The point at which this line cuts the axis is the *optical centre.* Rays passing through it suffer no angular deviation. The *principal focus* of a *convex* lens is a point on the principal axis to which

parallel incident rays are brought to a focus; and its distance from the lens is its *principal focal distance.*

When a luminous point beyond the principal focus sends rays to a convex lens the emergent rays converge to another point; the two points thus related are called *conjugate foci.* When rays diverge from a point whose distance is equal to, or greater than, the principal focus, the conjugate focus is *positive;* when the distance is less than the principal focus, the conjugate focus is *negative.*

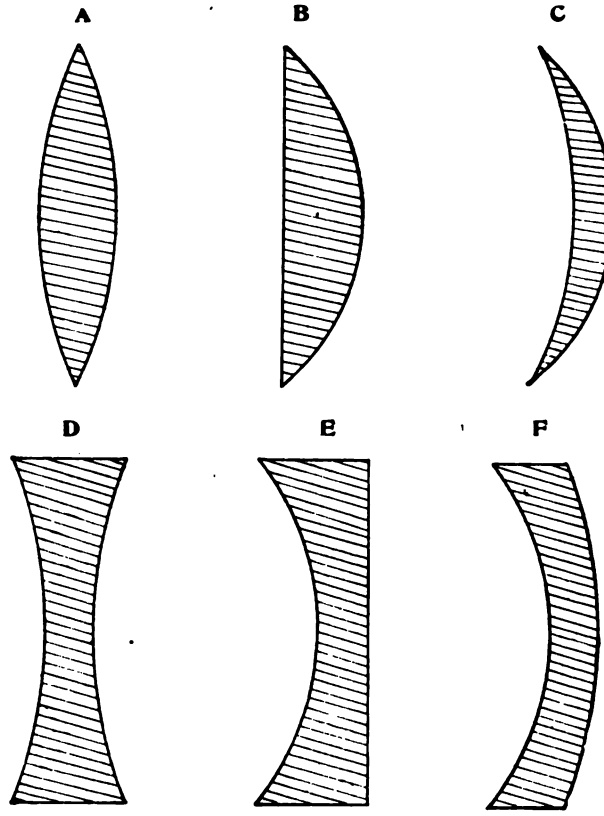


Fig. 389.—Profiles of lenses.

(A) Double convex
(B) Plano-convex
(C) Concavo-convex
} Converging lenses, thickest at optical centre.

(D) Double concave
(E) Plano-concave
(F) Convexo-concave
} Diverging lenses, thinnest at centre.

The *image* of an object placed nearer the lens than the principal focus is virtual, magnified, and erect. When the object is situated at a greater distance from the lens than its principal focus, the image is a real, inverted one. The *size* of the object and its image are proportional to their respec-

tive distances from the optical centre. The *position* of an image is found by drawing two lines from each extremity of an object, one passing through the optical centre of the lens and one parallel to the principal axis; the latter will be refracted to the principal focus, and where these two lines intersect the image will be formed.

The cardinal points of a *concave* lens are determined in the same way as those of the convex lens. All parallel rays falling upon a concave lens diverge after refraction and can never come to a focus on the far side of the lens, but if they are followed backward it will be seen that they come to a point between the lens and the object. This point is the *principal focus,* and, as the light does not really pass through it, it is called a *virtual* focus.

A *cylindric lens* is a lens one or both surfaces of which are segments of a cylinder, being composed of a series of prisms placed side by side. The resulting convergent points of every individual series composing the row must make a *line* of convergence.

Cylindric lenses may have any of the general forms of spheric lenses. If the series of prisms are arranged with their bases together, a *convex cylindric* lens is formed; or *concave* if arranged with apexes together.

The utmost refraction of a cylinder always occurs in the meridian at right angles to its axis. It acts only in one plane. To determine the axis of a cylindric lens, hold so as to see through it a part of some straight line, then turn the lens in its own plane until the part seen through it appears continuous with the parts above and below, the axis being then parallel to this line.

For sphero-cylindric combinations the *toric* lenses are desirable, as they increase the field of vision and reduce spheric aberration. The *toric* lens has the difference of refraction of the two principal meridians ground on one surface and the other surface ground concave, giving a periscopic sphero-cylindric lens much superior to the universal grindings.

Numbering of Lenses.—Lenses are numbered to indicate their focal distance, the unit being a lens having a focal distance of one metre and called a *dioptre,* the numbers expressing the refractive powers being multiples of this lens. A lens having a focal distance of one metre would have the sign 1.00 D. to represent it; a plus or positive sign is prefixed (+ 1 D.) if convex, and negative or minus (— 1 D.) if concave. Should the lens be a spheric one, the sign S. is placed between the character and strength sign. If it be a cylinder, the sign C. is substituted for the letter S. and the axis-angle is expressed by the abbreviation ax. with the degree of angle added (+ C. 1 D.; ax., 90°). The combination of a sphere and cylinder would be written, if convex, in the following manner: + S. 1 D. ⌒ + C. 1 D.; ax., 90°.

A lens of two dioptres' power (2 D.) focuses at one-half metre; a 4 D. lens at one-fourth metre. A lens that focuses at two metres is known as a 0.50 D. One at four metres' distance = 0.25 D.

To find the focal length of a lens in the dioptric system divide one

metre, or one hundred centimetres, by the number of dioptres; thus, the focal length of a lens of 4 D. is $^{100}/_{4} = 25$ centimetres.

The inch system was the old way of numbering lenses according to their radii of curvature expressed in inches. The unit 1 was a lens with a focus of about one inch. The successive strengths were expressed by fractional parts of 1; as a lens of 4-inch focus was expressed as $^{1}/_{4}$, and of 40-inch, as $^{1}/_{40}$.

To convert any strength of dioptre lens into an equivalent one of the old system, divide the dioptric number into 40 (there are 39.37 inches in a metre), and the inches of focal distance will be obtained. Or having the number of inches of focal distance of a lens and dividing it into 40 we get the number of the lens in the dioptric system.

With the dioptric system the calculation necessary for any combination of lens-power is very easy. As example, a + S. 2 D. added to + S. 4 D. gives a lens of + S. 6 D. Or, with unlike combinations, a + S. 4 D. and a — S. 1 D. added together give a resultant of + S. 3 D.

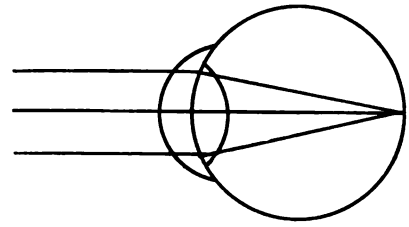


Fig. 390.—Emmetropic eye.

Similarly formed lenses produce results equal to their added powers, while dissimilarly formed lenses give results equal to their differences. The same rule holds good for cylinders in similar axes, while prisms increase in strength when placed base to base, and decrease in strength when placed edge to base.

EMMETROPIA.

Emmetropia is that condition in which the eye, in a state of rest, focuses parallel rays of light exactly on the layer of rods and cones of the retina (Fig. 390). While this is the mathematical definition of emmetropia, it is impossible to consider the functions of the visual organ as dominated by any fixed mathematical laws. Such an eye cannot be termed a *normal* eye, for it may easily be abnormal or morbid and nevertheless be emmetropic. If the emmetropic eye be considered, as it scientifically should be, as a perfect visual mechanism, in which parallel rays of light are brought to a focus exactly on that part of the retina devoted to distinct vision when the eye is in a state of rest, it is doubtful that it has any existence at all. The emmetropic eye, as it is clinically seen at times, is but a temporary condition in what is known as *diminishing hypermetropia*—the transitional

stage between hypermetropia and myopia, a refractive ha
inflamed eye that needs treatment.

An eye is *normal* in which the structures are free
ciated with undisturbing physiologic action giving as ne
result for both near and far as possible. The size or sl
quence. If it be healthy and acting properly, it is nor

An eye in which the principal focus falls on the
its dimensions: the shorter the radius of curvature of t
lens, the closer the retina would have to be to the diop
versely, the longer the radius of curvature of the cornea
the retina must be from the dioptric surfaces. As ext
an emmetropic eye with a corneal radius of 8.04 milli
of 24.94 millimetres, or one with a corneal radius of 6.9
visual axis of 20.95 millimetres. Arlt adopts a corneal
metres and an axis of 24 millimetres as the average stanc
In the newborn babe the average diameter in the visual
metres, the average horizontal diameter is 17.2 millimet
vertical diameter is 16.38 millimetres.

Theoretically, the emmetropic eye is probably the be
fort, and should always be sought for, even though it i
point that cannot be gained. While it may be a bre
restore the eye to a condition of emmetropia by suitabl
tion artificially obtained is not always the best for th
organ. A perfect vision obtained by neutralizing len
with increased physiologic action that is detrimental to
ical material.

AMETROPIA.

When the dioptric surfaces do not perfectly focu
through them, or when they do so focus it, but the ret
where, the eye is said to be *ametropic*. Alteration in
diameter of the eye by which rays do not cross one anoth
focus falls either in front or back of the retina, is known
Error in the curvature of its lenses is termed *meridional*
of refractive strength dependent on peculiarities in tl
dioptric media themselves is designated as *medial am*
principal focus falls behind the retina (*H*, Fig. 391), the
as *hypermetropia*. When the principal focus falls in
(*M*, Fig. 391), it is termed *myopia*. When there is more
focus, it is termed *astigmatism*.

HYPERMETROPIA.

Definition.—Hypermetropia is that form of ametro
in a state of rest focuses parallel rays of light behind tl

The refraction power of the eye is so low or its axis is so short that, when the eye is in a state of rest, parallel rays are not united upon the retina, but behind it, and only convergent rays are brought to a focus upon that membrane. Inasmuch as all objects reflect either divergent or parallel rays, such an eye is unable in a state of rest to see distinctly unless it can add to its refractive power. This is accomplished by using its accommodation. It can then cause its lens to become more convex and thus refract the rays more strongly, thereby causing the principal focus to be thrown on the retina (Fig. 393). This additional convexity takes away a portion of the

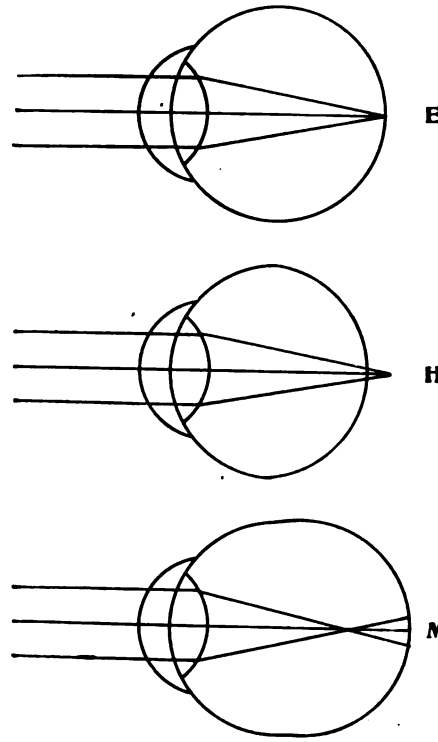


Fig. 391.—Refraction of parallel rays of light in emmetropia *(E)*, hypermetropia *(H)*, and myopia *(M)*.

accommodation power merely to preserve proper distant vision, leaving more or less of a minimum for purposes of accommodation. The result is that the near point will be farther removed from the eye; there will be a weakening of the dynamic or changeable power. Thus in Fig. 394 the parallel dotted rays have been sufficiently converged to be received upon the retina by the additional action of the lens, as shown by the dotted curve. A certain amount of lens-action has been lost in this procedure. The remaining portion is useful only for accommodation. There being less dynamic play of lens-strength, the near point will be situated at a much greater distance than in either emmetropia or myopia.

Etiology.—The hypermetropic construction of the eyeball is congenital and often is hereditary. It is generally an imperfectly developed eye, the expansion of the retina is less, and there is a smaller optic nerve with a smaller number of fibres. The normal eyeball at birth is smaller in all the axes, as has been previously shown, and there is an hypermetropia of about 3 dioptres, which is diminished by about one-third or one-half, not only by further development of the eye, but in many cases by pathologic change. In addition to the ordinary congenital abnormality of having too short a

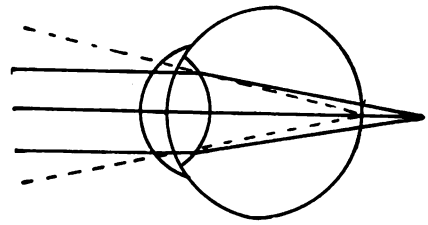


Fig. 392.—Inability to focus parallel rays of light on the retina of the hypermetropic eye.

visual axis or too weak a dioptric apparatus, the shortening of the antero-posterior diameter of even an emmetropic or a myopic globe so as to produce a marked hypermetropia by pressure has been attributed to augmentation of the retrobulbar contents of the orbit from vascular change, excessive adipose tissue, tumors, etc. Forward displacement of the retina in the macular region by localized chorioidal disturbance or submacular exudate may so lessen the power of the refractive constants in the visual axis as to increase

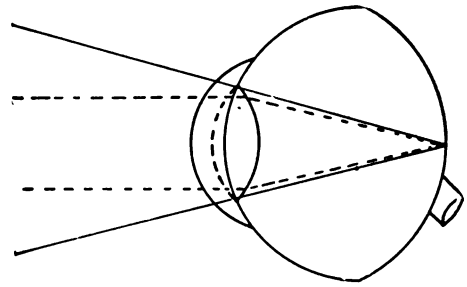


Fig. 393.—Accommodation is necessary to bring parallel rays on the retina of the hypermetropic eye.

an already existing hypermetropia, or reduce a myopia or emmetropia to an hypermetropia. Flattening of the cornea from traumatism and inflammation, and lid-pressure on the anterior face of the eyeball, are said to be the causes. Aphakia, or absence of the lens, is one of the most frequent causes of acquired hypermetropia.

Pathology.—In many cases of hypermetropia the orbits are more shallow than in emmetropia or in myopia, the margins are flattened and widely separated, the eyes are wide apart, and the nasal bone is depressed.

There is congestion of the lids and conjunctivæ, which may become aggravated into formation of pustules along the margin of the lids—in the majority of instances associated with microbic invasion. Hordeola and Meibomian-gland cysts are frequently seen. There is hypertrophy of the internal muscles. The sclerotic is thickened and dense. The circular fibres of the ciliary muscle are generally hypertrophied. In the higher degree of hypermetropia the hypertrophy is so great that there is either a right angle or an obtuse angle formed at the junction of the anterior and the external surfaces. This is less so in emmetropia. In high myopia the angle is often quite acute. The extra exertion put upon the ciliary muscle to overcome the optical defect causes an increased flow of blood to the eye. The optic disc at times exhibits a low-grade inflammation. The lymph-channels of the main retinal vessels are often thickened and opaque, while the veins may be somewhat engorged. The retina and the chorioid are not infrequently congested, especially in the region of the macula lutea and of the optic disc. The fovea centralis is situated quite distant temporally from the outer edge of the optic-nerve head.

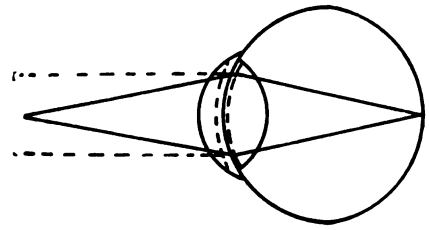


Fig. 394.—Additional accommodation power is necessary to bring an object at the near point upon the retina of an hypermetropic eye.

Symptoms.—The symptoms are (1) subjective and (2) objective.

1. Subjective Symptoms.—Low and medium grades of hypermetropia are normal in the eyes of all the human race which are not excessively and constantly used for near work. It is only when the vision becomes indistinct or painful because of diminished power of accommodation, or higher hypermetropia, that the patient seeks relief of the physician, who should be thoroughly familiar with those symptoms and their causes.

In the lower grades of hypermetropia, especially in the middle-aged or in those using their eyes for observation of minute objects at close distances many hours daily, the patient complains of the eyes tiring easily. They finally get sleepy, their lids become heavy, their eyes itch or burn, and the print begins to blur during continued near work. They are compelled to stop, and a sense of relief is felt by pressing the eyes and forehead with the hands. Work is resumed, but the symptoms recur. This is repeated until the accommodation becomes exhausted *(accommodative asthenopia)* and the patient is forced to discontinue work. The symptoms are always aggravated by artificial light. If work is persisted in, headache over the eyebrows and in the temples generally appears.

In the higher degrees of hypermetropia the ciliary muscle is so little able persistently to combat the defect that any effort at continued near work is relinquished. In healthy young people the eye is able temporarily to overcome the error by severe and persistent effort, but the hours of work cannot be maintained continuously. The effort and strain on the part of the ciliary muscle are shown by pain in the eye or temple and forehead. In aggravated cases the pain shoots through the top of the head to the occipital region. Often the neuralgia is so intense that any change of tension in the ciliary muscle becomes painful, and reflex disturbances, usually consisting in sensations of dizziness and nausea, are developed. These symptoms are excited by work requiring rapid change of focus, as in bookkeepers, pianists who alternately glance at a sheet of music and the keyboard, or in those learning typewriting before the necessary manual movements have become sufficiently automatic not to require any fixity of gaze upon the variously placed characters. Less severe, but similar, symptoms occur in low grades of hypermetropia in persons who are convalescent from acute disease, or in those who have enfeebled or broken-down nervous systems, and in women with pelvic disease, uterine displacement, etc., especially marked at the menopause, and during irregular menstruation, appearing after prolonged periods of abuse of the eyes, or during mental anxiety or undue fatigue. The symptoms are most severe soon after rising. The patient complains of pain in the back and top of the head, accompanied by great discomfort and dull pain deep in the orbits immediately back of the eyes. The ready fatigue of the eyes on very slight use and the neuralgia, discomfort, and blur caused by slight and moderate degrees of hypermetropia may also be very early symptoms of posterior spinal sclerosis.

In the hypermetropic eye the acuteness of vision is often diminished. In the slight degrees there is seldom any difference to be observed, but in the higher degrees of hypermetropia among the young, and in nearly every case in subjects who are beyond forty-five or fifty years, visual acuity for both distant and near work is reduced below the standard.

On account of the shorter distance between the nodal point and the retina, the retinal images are smaller than in the emmetropic eye, and, even after the correcting convex spheric lenses are placed properly before the eyes so that the retinal images may be considered to attain the normal magnitude, the acuteness of vision in many cases does not reach the normal, probably because the smaller optic nerve does not possess the same number of nerve-fibres and the smaller surface of the retina does not contain the same number of percipient elements as the emmetropic eye.

In the very highest degrees of hypermetropia the retinal images become so small that the patients bring objects close to their eyes in order to make the images larger. At the same time they nip their eyelids so as to reduce the diffusion circles. This symptom may be mistaken for myopia, but can be distinguished by the want of uniformity in the distance at which the patient places his book. He holds the book far off, then again closer, and

even if the print is large and distinct he is often unable to read fluently at any distance.

What properly characterizes the vision of the hypermetrope is not the diminished acuity of vision, but the recession of the near point, which is especially pronounced during monocular vision. As was previously explained, the emmetropic person relaxes his power of accommodation as much as possible and then sees acutely at any infinite distance; but the hypermetropic individual in order to see at a distance must bring his power of accommodation into action; consequently there is less power left to focus near objects, the lens reaches its maximum tension earliest, and for that reason the patient is forced to hold objects farther away from the eye than the emmetropic. This constant contraction of the ciliary muscle frequently produces a *spasm of the accommodation*, especially in neurasthenic individuals, giving rise to annoying blurring of distance vision; both the near and the far point are brought nearer the eye, and vision is improved by concave lenses, which, of course, should never be ordered, as they would only increase the trouble. The condition simulates myopia, and might be mistaken for such by a careless observer unless the eye is brought thoroughly under the influence of a cycloplegic.

The concealing power of the ciliary muscle and lens has given rise to the description of three varieties or types of hypermetropia in ophthalmic nomenclature. When the defect is completely concealed by the accommodation in both distance and near vision it is known as *latent hypermetropia*, and has the symbol Hl. That portion which remains uncorrected and is exposed, giving rise to painful or indistinct vision of near objects or misty outlines of distant ones, is designated *manifest hypermetropia*, expressed by the abbreviation Hm. In other words, it is that part of the defect that can be corrected by convex lenses without the use of a cycloplegic, whereas the latent can be developed only by completely paralyzing the ciliary muscle. The entire amount of the error obtained by adding together the *manifest* and *latent* is termed *total hypermetropia*—expressed by the abbreviation Ht.

Manifest hypermetropia is further divided into *facultative hypermetropia* (Hf), which exists when objects can be seen accurately at infinity with and without convex lenses, and without any convergence being necessary; *relative hypermetropia* (Hr), in which there is only sufficient accommodation to neutralize the defect by an undue effort at convergence; and *absolute hypermetropia* (Ha), when, even with the strongest convergence of the visual lenses, accommodation for near and far vision is impossible—objects being seen indistinctly at any distance.

2. Objective Symptoms.—The physiognomy is often characteristic of hypermetropia, especially in the higher degrees. The eyelids are flat and broad; the eyes are small, widely separated, sometimes deep-set, and at other times they are superficial. By some the orbits are said to be shallow, with flattened and widely separated margins; others describe them as being both

high and narrow. The whole face is flattened; there is little rounding in the cheeks. The nose is but slightly prominent, with depressed nasal bridge. Not infrequently there is thickening of the conjunctiva, with stillicidium and epiphora, and a predisposition to herpetic disease of the cornea. Immediately around the cornea the sclerotic has a flat, slightly curved appearance. At the equator the eyeball is sharply curved and bulging. The cornea does not share in the imperfect development of the remaining structures, which prevents the hypermetropic eye being classed as true microphthalmos. It has the appearance of being more convex, due solely to the shallow anterior chamber and small pupil.

In hypermetropia there is an apparent divergent strabismus when looking at a distance, due to the increased width of *angle alpha*—the angle formed by the visual line, which proceeds from an extraneous object directly through the nodal point to the macula lutea of the eye, and a line through the axis of the cornea, on the horizontal plane. The visual line in emmetropia passes to the nasal side of the axis of the cornea. The macula lutea in the hypermetropic eye is situated farther to the temporal side of the optic-nerve head than in emmetropia. Consequently the visual line cuts the cornea farther to the nasal side, increasing the *angle alpha* in high-grade cases to nearly double the width of that which is found in emmetropia; so that in hypermetropia in order to give a parallel direction to the visual line an extraordinary divergence of the visual axes is necessary, and, if in distance vision the necessary divergence of the corneal axes is insufficient, the convergence in looking at near objects will be relatively too great—a condition that facilitates the development of *convergent strabismus*.

In hypermetropia the patient must make an effort of accommodation to remedy the insufficiency of his static refraction. For every increment of nerve-force sent to the accommodation an equal amount is sent to the convergence, and, as an equal amount of nerve-force is sent to the accommodation and convergence of the two eyes, there must be an excess of convergence produced, which is still further exaggerated when the patient looks at a near object. This effort on the part of accommodation and convergence being in constant use in order for the hypermetrope to have clear vision, the habit of convergence is formed, which causes an increase in the development of the converging muscle with a corresponding weakening of the abductors, and the convergence becomes pathologic, but does not manifest itself at the same time in both eyes.

The nerve-force necessary to keep the eye directed upon the object is equally divided between the muscles of the two eyes, but in one it maintains the equilibrium between abductor and adductor muscles, while in the other, in which the sight is more feeble and the image more easily suppressed, it goes entirely to the adductor, forming an angle double that which would be necessary if the convergence were symmetrical. Therefore we ordinarily see but one eye "squinting." It usually develops early in life, in poorly nourished children, when they begin to fix their attention upon

objects shown them or upon first entering school. It generally begins by being *periodic,* or *alternating;* later it becomes *permanent* and localized to one eye, generally the weaker, although it may alternate for years. It occurs principally in the medium degrees of hypermetropia. The higher degrees do not "squint," because good vision is out of the question under any circumstance.

Not all hypermetropes have strabismus, because of the innate tendency to binocular vision and the changeable relations between accommodation and convergence. Convergent strabismus from hypermetropia tends gradually to diminish as the patient grows older and often passes off spontaneously; so that it is rare after the age of forty. Of course, there are other causes of convergent strabismus which are discussed elsewhere in this book.

Ophthalmoscopic Appearance.—In examining an hypermetropic eye with the ophthalmoscope by the direct method, the fundus appears brilliantly illuminated; the ophthalmoscopic field is greatly enlarged. The low degree of magnifying power causes the optic disc to appear smaller than in emmetropia, and if the observer be emmetropic and his accommodation be relaxed, it will require a convex lens revolved before the sight-hole to render the image distinct. This is all that will be observed in the very low grades of hypermetropia.

In the moderate degrees, in addition to the appearance mentioned, there is increased redness of the optic disc, congestion, and tortuosity of the retinal veins, which is most marked near the disc. The thickening of the lymph-channels of the main retinal vessels produces a bright yellowish-white reflex, shifting with the motion of the mirror. The epithelium of the chorioid presents a smooth, hazy appearance. Frequently there is congestion of the chorioid and retina, especially in the region of the optic disc and the macula lutea.

In the higher degrees of hypermetropia the media are clear and the disc, retina, and chorioid appear nearly normal, because the patient cannot overcome his defect by increasing the accommodation to secure distinct vision. Therefore, with less physiologic action, less blood is sent to the eye, and the eye does not present the congested ophthalmoscopic picture of the lower grades, in which the patient can secure distinct vision by increasing the accommodation, thereby inviting an increase of blood to the eye.

Diagnosis.—The history of headaches coming on during or after the use of the eyes; the usually slight impairment of distance vision, which may be improved by the addition of convex lenses; the removal of the near point farther from the eye than is proper for the age; the fact that the patient can read fine print through a convex lens at a greater distance than its principal focus; the characteristic mold of the face—the thickened and irritated conjunctiva, the small eyeball, sharply curved at the equator; possibly a convergent strabismus; the narrow anterior chamber; the small pupil; the great width of the ophthalmoscopic field and small image in

the direct method; the fundus-image test, and the fundus-reflex test (described elsewhere), all determine the presence of hypermetropia.

Treatment.—It must be remembered that hypermetropia of low grade is the normal state in the eyes of all the human race which are not excessively and constantly used for near work, and that the proportion of hypermetropes is much greater among children than among adults. The eye participates in the general development and there is a gradual diminution of the hypermetropia during the period when the entire organism is growing and acquiring its definitive form. Therefore hypermetropia should receive treatment only when it produces asthenopia or insufficiency of sight.

As we possess no radical cure for hypermetropia, we must resort to the most effective palliative remedy—convex lenses. Such glasses, by adding their refractive power to that of the eye, can supply any deficit in the latter and correct no matter what degree of hypermetropia.

Theoretically it would appear right to neutralize the hypermetropia by a convex lens, and thus change the eye into an emmetropic one, this lens forming, so to speak, an integral part of the eye. But in practice we find that this is undesirable, and that such patients will reject such glasses so long as they can see distinctly at a distance without asthenopia. This arises from the fact that eyes which since childhood have been accustomed to strain their accommodation and convergence in the effort to see distinctly, and have thus acquired hypertrophy of the ciliary muscles and of the internal recti, are unable at once to reverse their habits of work with comfort.

In addition, it must be remembered that any glass, no matter how well fitted to the eye and face, is a nuisance by giving rise to circles of diffusion which arise from reflected images of brightly illuminated objects striking its posterior surface, thus making the edges and rim of the glasses unpleasantly apparent. Disagreeable feelings in the nose, temples, and ears are also often occasioned by the weight of the glasses and the pressure of their frames. If distance vision is made much more distinct with the glasses, or if headache and eyestrain are relieved by their use, the advantages resulting from their employment lead the patient to habituate himself to them and to neglect the inconveniences. But we should abstain from giving spectacles except in cases of absolute necessity.

When spectacles are found necessary they should be ordered and adjusted by a physician. If opticians would devote their energies to grinding lenses properly and perfecting optical instruments, they would do more toward furthering the service of medicine, and relieving the suffering of humanity than by attempting to do something for which they are ill or not at all prepared. Too much cannot be said against the pernicious habit of opticians ordering glasses, which are as much a medicine for the diseased and complaining eye as is digitalis for a diseased heart. The pharmacist is far better prepared to administer the digitalis than is the optician to order glasses.

The eye must be regarded, not as a separate organ, but as a highly specialized part of the whole body. The ordering of glasses does not consist merely in placing lenses before the eye and ordering that which gives the best vision. The choice of glasses is a delicate operation; he alone is successful in it who, to a perfect theoretic acquaintance with the subject, adds the intelligent observation of each patient. It does not suffice to know the action of lenses and the workings of the visual organ. He must consider the state of accommodation and refraction, that of the muscles of the patient's eyes, the particular purpose of his wearing glasses, his peculiar habits, and the constitutional state of the patient.

Persons who are convalescent from acute disease, or those who have feeble health and impaired nutrition, as well as those who have hyperesthesia of the nervous system, suffer acutely from low grades of hypermetropia which would produce no symptoms of discomfort at the same time of life were they in robust health.

In the hyperesthetic state preceding central nerve degeneration, and in the various disturbances of health accompanying uterine or ovarian disease, adolescence, or the change of life, we frequently see that the correction of hypermetropia of even 0.25 D., or equivalent change in glasses already worn, relieves headache and nervousness, and enables the patient to perform his daily task with less discomfort. Again, persons who, in an enfeebled state of health, have found spectacles absolutely necessary for comfort, can perhaps lay them aside when they have regained their accustomed health.

As can be well seen, it is impossible to make rules for the treatment of refractive errors which may be blindly followed, relieving the oculist from the necessity of reflecting and combining for himself.

A correction should be prescribed in all cases of marked local vascular disturbance or where there is asthenopia. To ascertain this condition in young people, it is necessary to obtain the absolute amount of refractive error. This is done by placing the ciliary muscles under the full influence of a cycloplegic. Without such drugs it is impossible, except in the middle-aged and in the old, to ascertain the total hypermetropia, either by test glasses, by the ophthalmoscope, or by the shadow test.

Having ascertained the total amount of hypermetropia by the various methods described elsewhere in this book, the question is: what shall we order? The full correction placed before the eye while yet under complete influence of the cycloplegic gives distinct distance vision, but the same correction placed before the eye after the effects of the drug have disappeared will possibly not give anywhere near normal distance vision, and all distant objects will appear hazy, which clears entirely upon removing the correction, and yet if no correction is worn the asthenopia and vascular disturbance return. What is to be done?

This may be overcome by ordering the full correction to be worn constantly while the eyes are still under the effect of the cycloplegic, and, if this correction produces indistinct distance vision after the eye has regained

its normal condition, the glass may be weakened until full acuity of vision is obtained. Or we may first allow the eyes to regain their full power of accommodation; the full correction is then placed before the eyes, and, if full acuity of vision is obtained, it may be ordered. But usually the vision is reduced considerably. The patient is then requested to read as much as possible on the test-card, the strength of the glasses being gradually weakened until full acuity is obtained, and the strongest glass that gives full acuity of vision is ordered. The latter method should be followed whenever possible, as the results are more certain.

The principal objection to ordering from the post-cycloplegic examination is the delay to the patient in securing his glasses. This is an important consideration to most patients, but if one of the more rapidly acting cycloplegics be used, such as scopolamin,—which paralyzes the accommodation for ninety-six hours,—this objection is overcome, and, furthermore, if it should be necessary for the patient to use his eyes occasionally during this time, he can be supplied with a pair of + 3.00 or + 4.00 spheres.

Another objection to this method is that the total H is uncorrected. But it must be remembered that it is not always desirable or even safe to convert the eye into an emmetropic condition, and that the acquired hypertrophy of the ciliary muscles and of the internal recti cannot be lost at once. Consequently, when the full correction is placed before the eyes, there is an annoying blurring of distance vision, with headaches and general discomfort. It is true this may be overcome by using a very weak solution of atropin daily until unpleasant symptoms have disappeared, but few patients will submit to being deprived of their near vision for any considerable length of time. Of course, if there is any inflammatory condition within the eye caused by the H, atropin should be used until such condition has subsided before any correction is made, the tension being watched constantly.

Ordering glasses for the manifest error only, without any knowledge of the total H, is not to be thought of except for adults in whom the use of a cycloplegic to allow the immediate estimation of the total H is inconvenient or impossible. It is unscientific and harmful to the patient. The same may be said of arbitrarily deducting a certain amount from the total H.

As has been said before, there can be no rule to follow blindly in all cases. But the writer believes the method that will give greatest satisfaction to the patient is to make a careful estimation of the total H, and, after the accommodation has returned, place the full correction before the eyes and direct the patient to begin with the large letters and read slowly down the card while the correction is lessened 0.25 D. each time, forcing the patient to read the lowest line possible at each reduction, until full acuity of vision is obtained, ordering the strongest glass which gives such vision. If this glass is very much weaker than the total H, it should be worn only for distance, and the full correction prescribed for all near work. In ordering full correction, it should be remembered that, while rays of light coming

from a point five or six metres distant may be considered practically parallel, strictly speaking they are not, and a glass that gives fullest acuity of vision at that distance will not focus parallel rays. Therefore it does not represent the total H, but an overcorrection of $^1/_5$ to $^1/_4$ D. Consequently that glass which gives the best vision at five or six metres should be weakened by $^1/_5$ or $^1/_6$ D.

If there is insufficiency of convergence *(exophoria)* present, full correction is contra-indicated, whereas if there be *esophoria,* full correction will be worn with greater comfort.

If the glass correcting the total hypermetropia be placed before the eye while the accommodation is paralyzed, and a + 4 D. sphere is placed in front of that, the patient should be able to read small type distinctly at twenty-five centimetres—the focal distance of the lens. If the patient reads best at a point beyond this, the hypermetropia is not fully corrected, whereas if it is read nearer than twenty-five centimetres, the hypermetropia is overcorrected.

Convergent strabismus caused by hypermetropia, if in a child too young to wear glasses, may be treated by instillation of a weak solution of atropin

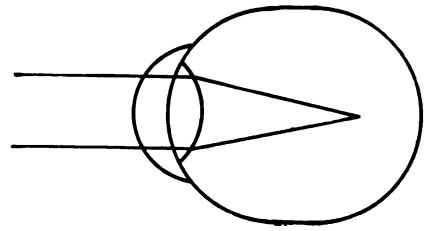


Fig. 395.—Illustrating the focus of parallel rays of light in the myopic eye.

in both eyes ($^1/_2$ to 1 grain to the ounce), and the eyes protected from light by a veil or shade. As soon as the age of the child permits, he should be given glasses that correct the full amount of H, and advised to use the eyes for near work not more than a few hours each day, with frequent intermission. The cycloplegic may be gradually withdrawn until discontinued.

If the visual acuteness in the convergent eye is very slight, the refraction-error should be carefully corrected, and then the deviating eye should be given special training, while the healthy eye is excluded. The exercise should never be continued until fatigue is produced. In this manner the visual acuteness can be considerably augmented.

If the strabismus persists after the above treatment has been carefully followed, it then becomes a case suitable for one of the several operations described elsewhere in this book.

MYOPIA (HYPOMETROPIA).

Definition.—Myopia is that form of ametropia in which the eye in a state of rest focuses parallel rays of light in front of the retina (Fig. 395).

In the emmetropic eye in a state of rest parallel rays are brought to a focus directly on the retina. The refracting power of the eye and the length of the visual axis are equal and a perfect image is formed on the retina. In hypermetropia the eye when at rest focuses parallel rays of light behind the retina. There is an insufficiency of refracting material, or the eyeball is too short and only convergent rays can be brought to a focus on the retina when the eye is at rest. The far point is beyond infinity and the near point is farther from the eye than was proper for the age. In myopia, the eyeball being too

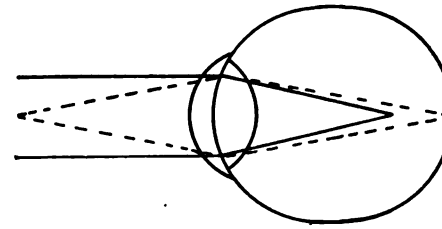


Fig. 396.—Only divergent rays are brought to a focus on the retina.

long or there being too much refracting material, the focus falls in front of the retina in the vitreous, and only diffusion circles are formed on the retina. Only divergent rays of light can be brought to a focus on the retina (Fig. 396). Consequently the far point is at a finite distance: *i.e.*, it is near the eye. The near point is also brought nearer the eye.

The myopic eye has just as much lens-power as the hypermetropic or emmetropic eye, but in the hypermetrope a certain amount is lost in maintaining distinct distance vision, thus having less power available for focus-

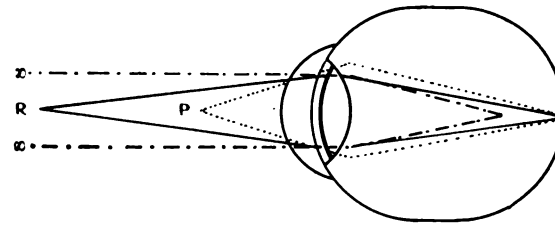


Fig. 397.—Parallel rays focused in the vitreous.
R, Far point. *P*, Near point.

ing near objects. The myope, not being compelled to use any of the power for distance vision, has that much more for focusing near objects, and in addition is assisted still further by the long visual axis, which enables him to see even closer than the emmetrope. Fig. 397 shows the far point and near point in myopia, with increase in the curvature of the lens for the near point.

Etiology.—Myopia is due to increase of the length of the visual axis by stretching of the eyeball at the posterior pole (*axial M.*). Myopia may be produced practically by excessive curvature of the cornea, as in staphy-

loma of the cornea and conic cornea, or in the early stage of cataract, when the lens swells and increases the curvature *(curvature M.)*, or the lens may be dislocated forward. It is also produced in spasm of the ciliary muscle; or, in fact, every time the lens accommodates for objects inside of the far point a practical myopia is produced. But these conditions do not cause a true myopia. The term myopia is applied only to the eyeball in which the increase of the length of the visual axis is due entirely to the stretching of the structures at the posterior pole.

There are numerous theories to account for the causation of myopia. It is believed by some to be due to a hyperinclusion of mesoblastic vitreous material into the secondary optic vesicle. By others it is believed that it depends upon the size and shape of the orbit—that the vertical diameter of the orbit is increased. By others the contrary condition is claimed—that in low orbits there is such a variation in the course and insertion of the superior oblique muscle, due to the low position of the trochlea, that it increases the amount of force, or compression, which this muscle exerts upon the eyeball. The dragging effect on the lamina cribrosa at the weakened portion of the eyeball,—the optic-nerve entrance,—associated with the increased compression, is certainly a contributing factor of the mechanics in the inflammatory and degenerative processes of myopia, when occupation necessitates continued vertical movements of the eyeball.

Others assert the cause to be due to compression of the globe by the external rectus muscle during excessive convergence made necessary by the short distance at which myopes must hold objects to observe clearly, the eyeball stretching posteriorly. Compresssion of the vorticose veins by the inferior oblique and external rectus muscles is also advanced as a solution of the etiology. Again, it is believed by some to be due to an inflammation of the chorioid and sclera at the posterior pole produced by constant congestion made necessary by occupation, in which the stooping position is assumed, or from compression of the jugular veins by bending over improperly adjusted desks or books, interfering with the return of blood from the eye, leading to congestion and subsequent inflammation.

Abnormalities of the retrobulbar portion of the optic nerve not permitting proper movement of the eyeball is yet another reason given. Race is also claimed as an etiologic factor. It is claimed that the broad, low face of the German contains the conditions for the production of myopia, but it seems more reasonable to presume it to be due to a previous want of proper correction of hypermetropic astigmatism. Since the disease has received the proper scientific investigation and more attention has been paid to hygiene in the school and home, it has rapidly decreased.

Myopia is far less prevalent in this country than in Europe, especially Germany, where it is a national defect. In this country more attention is given to out-of-door sports and exercise, and not so much to the close application of detail and great reverence for past work, that characterizes the sedentary, plodding German nation. Myopia is said to be unknown among

the Nubians, the Laplanders, the Patagonians, and the peasant classes of Europe. It is less frequent in the eastern portion of the United States than formerly, due, no doubt, to the improved methods of scientific investigation and the correction of minor degrees of hypermetropia and astigmatism. Jews are said to be more subject to myopia than Christians of the same social class. Since the abolition of slavery in this country and the introduction of higher educational methods, the student class of negroes has begun to develop myopia.

Myopia is very rarely congenital. That there is some hereditary tendency in the form of the myopic eye there can be no doubt. Just as the susceptibility to tuberculosis is passed from parent to child, so may the tendency to myopia be inherited. Myopia is as truly a disease as is tuberculosis, and, if an individual can be born with "something" in the economy or a lacking of "something" that predisposes that individual to the ravages of tuberculosis when exposed to the contagion, so might it be possible to inherit an eyeball the structure of which predisposes it to the development of myopia when placed under conducive conditions, of which the most important are improper illumination and hypermetropic astigmatism.

Almost all eyes are hypermetropic at birth and very few are free from astigmatism, and, comparing these with the number of myopes, it is evident that there is something necessary for the development of myopia other than hypermetropic astigmatism. But, given an eye predisposed to myopia and forced to work at a short distance under a poor light, the vision being already reduced because of the hypermetropic astigmatism, objects will be held closer than they should be in order to obtain a large retinal image, thereby making an undue effort at accommodation with exaggeration of convergence. Following the law of nutrition, an increased flow of blood is invited to the eye to carry nourishment to the constantly active ciliary muscle, producing first a congestion and later an inflammation of the ocular tunics. The increase of tension due to the increase of fluids within the eye presses upon the impoverished and diseased tissue. Not being able to withstand the strain, there is a consequent giving way in the direction of least resistance, which is in the posterior segment of the globe. The further progress is hastened by the formation of the orbit and the insertion of the extra-ocular muscles compressing the globe still further and causing the refraction to change from hypermetropic astigmatism to mixed astigmatism and finally over to myopia with astigmatism.

All children, especially improperly nourished children (both of rich and poor), are put to using their eyes for near work at an age when they should be having outdoor exercise and the eyes should be resting on distant objects. For this reason kindergarten work is harmful. It trains the mind by systematic play, but does so at the expense of the eyes, and it is quite likely that the beginning of myopia could be traced to kindergarten and primary-school work done under bad hygienic surroundings, combined with uncorrected hypermetropic astigmatism.

That school-life has harmful influences upon the eye is very clearly shown in the adjoining diagrams from statistics collected by Risley in Philadelphia schools and Erismann in St. Petersburg. In Fig. 398 it is seen that, at the beginning of school-life,—eight and a half years,—88.11 per cent. of the eyes examined were hypermetropic, with only 4.27 per cent. myopic. Then there is a gradual sinking of hypermetropia until the age of seventeen and a half years is reached, when it is present in only 66.84 per cent., and at the same age myopia has increased to 19.33 per cent. Fig. 399 shows the very rapid sinking of hypermetropia and rise in myopia as the pupil advances in school-life in Europe as compared with the United States. Cohn's statistics show the very rapid increase of myopia from 1.4 per cent. in the lower grades to 59.5 per cent. in the university.

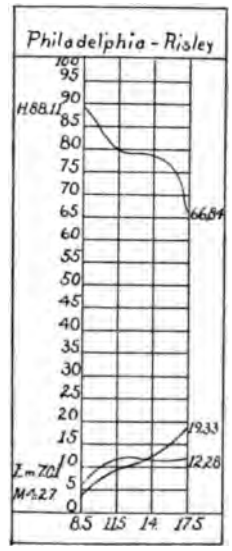


Fig. 398.

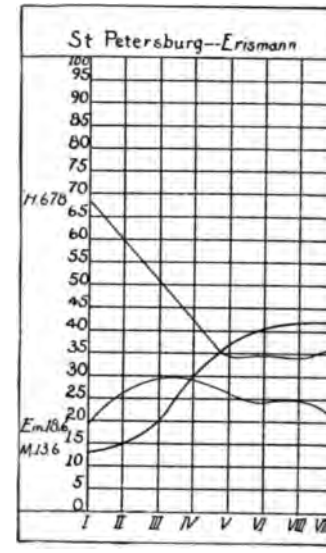


Fig. 399.

Study in itself is not the cause of the great increase in myopia as the pupil progresses. Potent factors are: Studying of books poorly printed, with small type, lines too close together and extending across the page; improperly adjusted desks and stools, poor illumination, faulty ventilation, too long hours of study with insufficient intervals, combined with uncorrected hypermetropic astigmatism. That bad hygiene in the school-room is not alone the cause of myopia is shown in the studies made by Derby, Cohn, and Yust, showing the steady increase of myopia in schools having perfect hygienic surroundings and where great attention is paid to physical culture. Almost all authorities agree that the perfect hygienic arrangement of the school-room alone will not prevent the development of myopia.

There are some who regard the change in refraction as a process of normal evolution, and claim the myopic eyeball is better suited to the needs of civilization than is either the emmetropic or hypermetropic globe. In

order to sustain this opinion it would be necessary to show that this change of refraction from hypermetropia to myopia does not interfere with the health and comfort of the eye. That quite the reverse of this is the truth is clearly demonstrated in Figs. 400 and 401, which show (in Fig. 400) the gradual increase in disease of the eyeball with the state of refraction passing from hypermetropia over to myopia, and (in Fig. 401) the increase of asthenopic symptoms as the refraction increases, which is quite sufficient to prove that the increase of refraction is not a physiologic process, but the result of diseased structure, and that, instead of its being a comfortable, the eye is a complaining organ.

Those living in cities are more susceptible to the development of myopia than inhabitants of country districts, where social and commercial competition is less marked. The unnecessary prolongation of the school-term, with

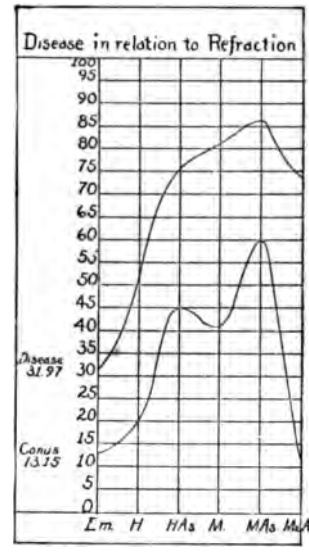


Fig. 400.

too short vacation, with the consequent enervation of the growing child, obtain in the city, as compared with limited term of school and extended vacations made necessary in the country. There the eye rests on objects at infinite distance, little exertion of accommodation and convergence being required even in the hypermetropic organ. The air is fresh, water pure, food clean and wholesome, and as a result there is an absence of the bespectacled, stoop-shouldered, narrow-chested, prematurely old children seen in all large commercial centres.

The susceptibility to myopia is most marked when both parents are myopic, less when the mother is affected, and least when the father alone is myopic. When both father and mother are affected, it is seldom that the children escape. It is claimed by some that the susceptibility is most frequently passed from mother to daughter.

Acquired myopia is said to begin from about the eighth year upward, but the process usually has its inception in a diminished hypermetropia with astigmatism, at a much earlier period, and ophthalmoscopic changes would undoubtedly be seen if looked for. During the period of adolescence myopia is almost always progressive. Puberty seems to affect the resisting power of the tissues, but, if the myopia does not attain a high degree during this period, it may become stationary when the body has completed its development and growth—about the age of twenty to twenty-five years. If the myopia reaches a high degree during youth, it is less likely to become sta-

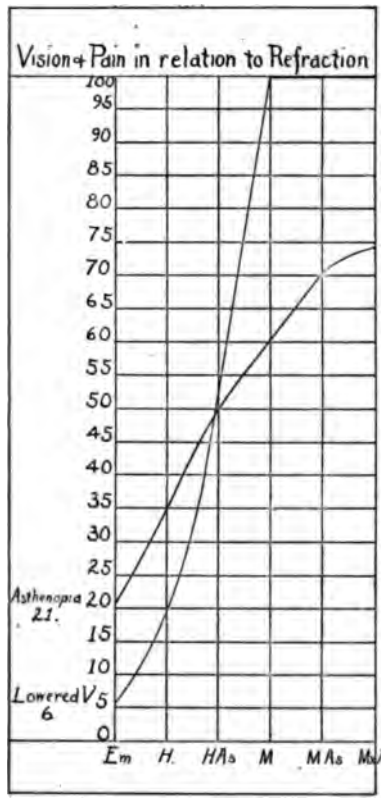


Fig. 401.

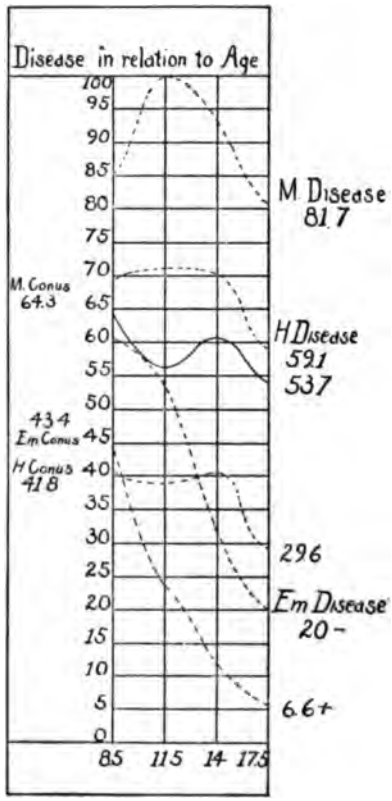


Fig. 402.

tionary. Vision will grow gradually less useful until the age of fifty or sixty years is reached, when the patient frequently becomes blind. The period from the fifteenth to the twenty-fifth year seems to be the time during which the myopia is most likely to progress both in degree and rapidity—the higher the degree, the more rapidly it progresses. Myopia seldom develops in an eye previously sound after the twentieth year. When there is only a trace of myopia present in youth, it almost always increases in degree, and the greatest care leads to nothing more than a limitation of the degree.

Myopia is seen most frequently in the more successful members of a community, and in those whose occupation requires close observation, such as compositors, brass-turners, etc. Although, frequently, high degrees of myopia rapidly progressive may occur in those whose eyes are never persistently applied for near work, some of the most malignant forms have been seen in sailors and laborers. Consequently myopia is not always a sign of superior mentality, and is not a characteristic of the student any more than of the artisan whose trade requires accurate near vision, if his work was begun early in life.

Pathology.—Myopes have on an average a less convex cornea than emmetropes. The anterior chamber is deeper. The pupil is wider, due to atrophy of the pectinate ligament. The crystalline lens is not more convex than in emmetropia. It is claimed that the lens is flatter. The circular fibres of the ciliary muscle are atrophied and are almost entirely wanting. The longitudinal fibres are greatly hypertrophied. With the increase in length of the myopic eyeball the vitreous humor must either increase in size or leave a space between it and the retina, which space soon becomes filled with serum. Frequently there is a softening of the posterior part of the vitreous, and a breaking down of its structures, with the formation of floating opacities. The vitreous may become detached.

In high degrees of myopia the drag upon the retina is so great that it is torn most frequently at the equator. The serum collected between the vitreous and the retina finds its way through the rent, and, passing down between the layer of rods and cones and the pigment layer, floats the retina forward (detachment of the retina).

There is a condition in the fundus of the eye of myopes to which the term *conus* is given, which presents the appearance ophthalmoscopically of a white crescent, usually situated to the temporal side of the disc, or it appears as a white ring surrounding the nerve-head. It is customary to attribute this to atrophic changes in the chorioid surrounding the disc, brought about by the congestion and inflammation of the chorioid and sclera, leading to bulging back of the sclera, known as posterior staphyloma. That this conus (white crescent, or ring) is not the result of inflammation with subsequent atrophic changes of the chorioid, as is generally accepted, has been abundantly proven by the researches of Schnabel and others. It has been shown that the annular conus is due to the fact that the optic foramen (sclero-chorioidal canal) and the part of the optic nerve surrounded by it are unusually large, while the intraretinal portion of the nerve (the papilla) is smaller in diameter, because the fibres lose their perineural sheaths; hence a white zone is seen between the edge of the papilla and the peripheral portion of the connective tissue or of the chorioidal ring. So the white color does not come from the exposed sclera, but from the lamina cribrosa of the optic nerve. The layer of pigment epithelium begins at this ring. It has not disappeared from the white zone, because it never existed in the part of the fundus in which the conus is

situated. When a posterior staphyloma (bulging back of the sclera) does coexist with conus, as is comparatively infrequent, the chorioid over the staphylomatous area is thinner than normal, but there is no marked circumscribed atrophy around the papilla.

The causation of the crescentic conus can be easily understood when it is remembered that the pial sheath of the optic nerve in every normal eye unites with the border of the elastic lamina of the chorioid, and, if this part of the chorioid does not extend to the edge of the chorioidal canal, the pial sheath in order to join it must extend across the anterior surface of the chorioid as far as the edge of the elastic lamina. Now, it has been shown that within the limits of the crescentic conus the elastic lamina and the chorio-capillaris are absent, and the posterior layers are thinner than normal. There is also very little, if any, pigment, and few or no vessels, while over the anterior surface of the conus is spread the pial sheath of the optic nerve, and in front of this lie the fibres of the optic nerve. Absence of the elastic lamina cannot be regarded as the result of atrophic change, because the pial sheath of the optic nerve stretches over the defect and unites with the elastic lamina at the periphery, just as in the normal eye. The anomalous position of the pial sheath and nerve-fibres form a constant anatomic feature of the crescentic conus; consequently the defect in the anterior layers of the chorioid must be regarded as the result of anomalous development, and not of disease. Every conus is congenital.

The anatomic foundation of the crescentic conus in the myopic eye with posterior staphyloma is the same as the crescentic conus in the myopic eye of average length and form. An annular conus cannot develop from a crescentic conus; neither can a crescentic one enlarge and become annular.

The posterior staphyloma is a bulging backward of the sclera, forming a low, circumscribed protuberance with a circular base, or, if the distension is great, a conic elevation, situated between the outer side of the optic nerve and the tendon of the inferior oblique muscle. In others it may include the sclera surrounding the optic nerve; so that, instead of lying to one side, the optic nerve may form the summit of the staphyloma. The length of an eyeball with posterior staphyloma exceeds that of the longest emmetropic eyeball known.

Schnabel has shown that posterior staphyloma occurs most extensively in eyes in which there is defective development of the chorioid, and an absence of the external fibre-layer of the sclera in the vicinity of the optic nerve, with resulting anomalous insertion of the dural sheath and consequent abnormal size and shape of the intervaginal space. He demonstrates that the increase in the intervaginal space is not due to the separation of the inner from the outer layers of the sclera; that the outer wall of the space is always covered by the arachnoid sheath, and is always surrounded by the two vaginal sheaths and the posterior surface of the inner layer of the sclera, just as in the normal eye, and never by the separated scleral lamellæ. If the enlargement were due to stretching of the sclera, it would

be most marked on the side of the optic nerve corresponding to the summit of the staphyloma and site of the crescentic conus; but the contrary is the rule, for the intervaginal space is much wider on the nasal than on the temporal side of the optic nerve. And, if the enlargement had been caused by the gradual distension of the sclera, the arachnoidal trabeculæ on the inner surface of the dural sheath would be correspondingly more attenuated than in the normal eye, but the thickness of the trabeculæ is much greater than normally. If the outer sheath had been forcibly separated from the inner it would be tense and its fasciculi would be stretched; instead of this the sheath is relaxed; its fasciculi are wavy and have the appearance of being too long. So Schnabel concludes that, "in view of the facts that posterior staphyloma is known to have existed in the early childhood of myopes of high degree, that no proof has yet been furnished that the sclera of eyes with such a condition ever possessed normal thickness and shape, that its development is not preceded by any disease of the eye, and, finally, that inflammation and increase of intra-ocular tension do not cause posterior staphyloma, but a different form of bulbar enlargement, it is certainly warrantable to conclude that the condition is an anomaly of development of the eye resulting from congenital peculiarities of texture in the parts of the chorioid and sclera surrounding the optic nerve." Consequently if posterior staphyloma is a malformation, and not the result of disease, it cannot be acquired by eyes with normal membranes. And every eye with the posterior staphyloma of Scarpa is myopic; but only a very small number of myopic eyes have this type of staphyloma. He also claims that eyes primarily emmetropic or hypermetropic are in no danger of becoming affected with retinochorioiditis of the macula in consequence of acquired myopia. Only eyes with posterior staphyloma resulting from congenital malformation have, in addition to excessive myopia, an especial predisposition to that grave disorder.

In extreme cases of myopia there may be disseminated chorioiditis and chorioidal and retinal hemorrhages, with detachment of the chorioid. The crystalline lens becomes translucent or opaque.

Symptoms.—Subjective Symptoms.—Persons myopic to the amount of 2 D. usually complain of not seeing distant objects clearly. In the higher degrees they can see objects distinctly only when held very close. They notice that the moon, the stars, or a street-light does not present a clear and well-defined outline. They appear as irregular, shining discs, which are much larger in diameter than they are to the normal eye, due to diffusion circles on the retina. The field of vision is always decreased, especially noticeable when wearing the correcting lens. There may be photophobia and photopsia. They complain of their eyes aching, and this is always aggravated by near work. This symptom is particularly troublesome if there is also astigmatism present.

The young myope will acquire distinct mental habits and peculiarities if the defect be not corrected with proper glasses. He will early learn to

avoid outdoor sports because of poor vision, and will cultivate a fondness for occupation suitable for his range. He will devote his time to constant study, winning the plaudits of his friends, which further increases the desire to attain higher ideals. He becomes retrospective and perverted in his taste. He will become brusque in his manner and unduly self-reliant. He has in general no correct idea of the impression which his person or his words make upon others, because he cannot appreciate the feelings and change of countenance of those he is addressing. Consequently he retains his original disposition, and is possessed of a peculiar freeness and too great self-confidence, or, what is still more rare, a more than ordinary self-consciousness, or bashfulness, is developed. Because of his superior knowledge gained by constant application to studies he arouses the jealousy and envy of his fellows, which in turn causes him to become rude, in order to hide a fear of each new face. He is uncongenial, and finally chooses some narrow groove having but few congenial spirits in it, and one that is constantly demanding increased study. Much more of what passes in the world escapes him than he is aware of, and with respect to a number of things his knowledge is less correct, because he fills up what is deficient through the operation of a brisk imagination. He is seldom practical and always is in search of the metaphysical. He sets a premium on physical deterioration, and possesses but little true intellectuality, his mentality being a mere precocity.

Objective Symptoms.—Myopes have a tendency to approach closer than is necessary to the objects, and thus, particularly in sedentary work, to assume a stooping position. They prefer to read small print, and accustom themselves to small handwriting, avoiding as much as possible long lines, because by so doing they need not move the eyes and head so much as when they have broad pages and large letters before them. A certain awkwardness is frequently manifested in their bearing and gait. The orbits are low, broad, and shallow in the young, increasing in height as the subject matures. During distant vision the eyelids are nipped together in order to see more distinctly by cutting off the diffusion circles. The brow is wrinkled. The nostrils are drawn up and the head is projected forward. They have a stupid appearance. The eyeballs are large and apparently protrude.

There may be an apparent convergent strabismus when looking at a distance in high degrees of myopia, due to the fact that the visual line passes through the cornea to the outer side of the optic axis, and the eye must converge in order to bring the visual line to intersect the object. Angle gamma becomes negative.

Myopia requires more convergence of the visual lines, because vision takes place close to the eye. Convergence is made difficult by the form of the eyeball impeding the movements, the small angles gamma and alpha, and the elongation of the muscles. The myopic eye, because of its static refraction, has no need of accommodation in order to see at a short distance; so the difficult convergence is not excited as in hypermetropia. Under these

circumstances binocular vision is either not developed o
in a rudimentary way. Consequently, when there is a l
vision and accommodation, the eyes almost always diver
sons myopia is the principal cause of divergent strabi
divergence is relative—that is, when looking at a dista
are properly directed; in close work only one eye is used
Later it becomes absolute: there is divergence of the
tant as well as in near vision.

In myopia the conjunctiva is usually congested.
ber is deep. The pupil is dilated and the iris is sluggis

Ophthalmoscopic Appearance.—Because of the
in myopia, only a small portion of the fundus is seen w
position of the ophthalmoscope. It appears highly m
illuminated. The optic nerve is apparently narrower
meridian and longer in the vertical than in the emmet
due to the disc being placed more to the inside, the perpe
face being directed more strongly outward, and, this
obliquely on the nerve through the pupil, it appears sh
zontal direction. In the young the nerve surface shows
There may be a conus,—a whitish area, crescentic in sh
temporal side, or annular in shape, entirely surrounding
bounded by a pigment-ring. In cases of high myopia
present (Fig. 2, Plate XX). Retinal and chorioidal cha
(Fig. 2, Plate XVII).

In advanced cases the retinal vessels appear dragged,
their course. The main trunks diverge more at right
there any change in the calibre of the vessels. There
irregular pigment splotches, white atrophic patches
chorioid, especially marked in the macular region. Th
epithelial pigment throughout the fundus. In extreme
retinal hemorrhages, disorganization of the vitreous
and chorioidal detachment. The lens may become opa

Diagnosis.—Myopia is indicated by the broad fac
eyeballs, divergent strabismus, indistinct distant visi
vision. The near point of accommodation is closer
emmetropia at the same age, and there is improvemer
by a concave lens. Letters of double size are not
distance. By means of the ophthalmoscope and the re
ence of myopia becomes apparent.

Treatment.—Myopia is a disease which can be pr
measure, or at least much can be done to arrest its pro

As myopia is produced principally during adolescen
by continuance of near work, it is necessary to keep th
health so that the eye-tissues can resist any reasonab
them. Children recovering from any of the exhaus

should not be made to perform the same tasks as those in good health. They should be given sufficient nutritious food, fresh air, and exercise as well as general tonics.

Those in feeble health or affected with chronic diseases should receive special attention. Young children should not have bright objects and toys held too near the eyes. They should not be sent to kindergarten at an age when they had better be enjoying the free air, playing games out of doors. Children are usually started to school entirely too early in life, often at four or five years of age. This period of life should be devoted solely to the building up of all the tissues. The child should not be subjected to the destructive process necessitated by the so-called "play studies" as pursued amid unhygienic surroundings.

If all children could remain at home until they are eight years old, and then be sent to a school where some attention is paid to the general hygiene, myopia would rapidly disappear.

In our schools attention should be directed toward securing proper lighting and ventilation. A northern exposure, with the light admitted from behind and over the pupil's left shoulder, is to be preferred. One square foot of glass to five square feet of floor surface is sufficient if not shadowed by trees or houses.

Stooping over work, with consequent damming of blood in the head, should be made unnecessary by having adjustable chairs and desks. The chairs should be just high enough to allow the feet to rest firmly on the floor. The desk-top should be sloping and arranged so that the body may be held erect, and the print or writing placed at a suitable distance from the eye.

The great amount of writing, not only in school, but even at home, as required in some localities, should be protested against. School is the place for study. Children should be taught orally as much as possible; when writing is necessary it should be with pen and black ink.

Text-books should be printed on unglazed paper. The type should be heavy faced and eight point or ten point in size. Two or two and one-half millimetres' space should be between lines, on a double-column page.

A systematic examination of the eyes of all children upon entering school should be insisted upon, and all refraction errors should be carefully estimated and corrected.

REMEDIAL.—If myopia becomes once established, we should endeavor to arrest its progress by the strictest care of the eyes and the employment of concave glasses, so as to render any necessary use more comfortable and less dangerous to the patient.

In all young people in whom the accommodation is still active a cycloplegic should be used sufficiently strong to produce absolute loss of accommodative power, and especially so if there should be present any retinal haze and chorioidal woolliness. Repeated instillations on successive days should be made until the congestion of the chorioid and retina is diminished. As

a rule, no correction should be made so long as there is congestion or inflammation of the eye.

A young subject, with good acuity of vision, a range of accommodation suitable to his age, and the balance of the external eye-muscles good, having a myopia under 5 D., may be allowed to wear the full correction habitually, both for near work and for distance.

When the patient is older and the grade of myopia is higher, together with lowered acuteness of vision, the full correction should be lowered; or we should allow him to read uncorrected at his far point, wearing full correction only for distance. If his occupation makes it necessary to see distinctly objects lying farther off than his uncorrected far point, a partial correction should be given. In these cases the glass for near work is determined by finding the distance at which the work is to be done, and subtracting that number of dioptres from the strength of the distance glass. For example, supposing a myope of 4 D. wishes to see clearly at a distance of one metre (4 D. — 1 D. = 3 D.), a 3 D. glass would be required.

In very high grades of myopia, with marked diminution of acuity of vision, it is best for the patient to relinquish all attempts at binocular vision with reading glasses and to use the eyes alternately.

If there is weakness of the internal recti muscles amounting to ten or twelve degrees for distance, and a corresponding larger amount for near work, great relief can sometimes be obtained by dividing one or both of the externi. Smaller degrees of insufficiency are satisfactorily treated by decentring the glasses, so that their centres shall be farther apart than the centres of the pupils, that they may act as a prism with the base inward.

When the myopia is 15 D. or more, the crystalline lens is sometimes removed, in appropriate cases, with improvement in vision and increase in the distance at which the eyes can be used for near work. Bruns, of New Orleans, performs discission of the lens in young subjects who have 12 or 13 D. of myopia.

ASTIGMATISM.

Definition.—Astigmatism is that form of ametropia in which rays from one luminous point do not again unite into one point, or focus, on the retina.

Defects in the antero-posterior diameter of the eyeball have been studied under the subjects of hypermetropia and myopia: hypermetropia, when the axis of the eyeball was too short; myopia, the axis being too long, both pertaining to the length of the eyeball. The defects in the dioptric system of the eye are: spheric aberration, chromatic aberration, and irregularity of radius of curvature.

Spheric Aberration is due to the rays passing through the edge of a convex lens and those passing through the central portion not having the same focus. There is one principal focus for the more bent rays, those passing through the periphery, and one for the less bent rays, or those passing through the central portion. This defect is almost corrected in the eye by

the iris, which cuts off the most peripheral rays, and also by the increase in density and curvature from the periphery toward the centre of the crystalline lens.

CHROMATIC ABERRATION.—In vacuum all kinds of light appear to travel at the same rate; but in most dioptric media they are unequally retarded. Commonly the light with short wave-lengths (near the violet end of the spectrum) is most retarded and most refracted. The dioptric system

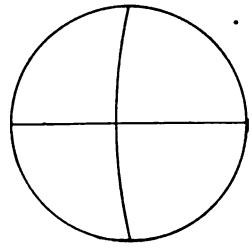


Fig. 403.—The cornea in emmetropia.

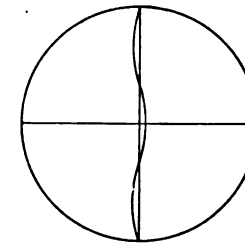


Fig. 404.—Irregular astigmatism.

of the eye also necessarily presents this chromatic aberration. Ordinarily, however, it scarcely interferes with the sharpness of vision.

IRREGULARITIES OF RADIUS OF CURVATURE.—It is with irregularities of radius of curvature of the lenses of the eye we are most interested at present.

In studying hypermetropia and myopia it was assumed that the dioptric surfaces of the eye curved equally in all parts and in all directions—

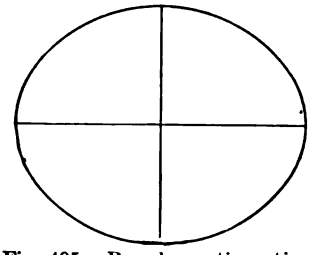


Fig. 405.—Regular astigmatism.

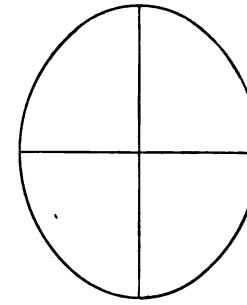


Fig. 406.—Regular astigmatism.

that they were small portions of spheric surfaces. But in reality they are not spheric in form. When even normal eyes are carefully examined, it will be found that the vertical meridian is usually more sharply curved than the horizontal. And the part of the dioptric surface in which this anomaly of curvature is most marked and constant is the cornea, producing regular astigmatism. In other words, the cornea, instead of being equally curved in all meridians, is more sharply curved in the vertical meridian and less curved in the horizontal. The difference in curvature is in different meridians (Fig. 405).

In irregular astigmatism there is a difference in curvature in different parts of the same meridian (Fig. 404). The seat of irregular astigmatism is usually in the lens. It is present in all human eyes, causing points or discs of light to appear to have irregular projections from them—to look "star-shaped." When it is in the cornea, it is generally the result either of irregular contraction of cicatrizations from previous ulceration or of softening and stretching from some pathologic process, such as conic cornea.

The seat of regular astigmatism is in the cornea, which has one meridian most sharply curved and another meridian at right angles to this with the longest radius of curvature. When the most sharply curved meridian, or meridian of greatest refraction, is vertical, or nearly so, it is termed *astigmatism with the rule* (Fig. 405). When the meridian of greatest refraction—most sharply curved—is in the horizontal direction, or nearly so, it is known as *astigmatism against the rule* (Fig. 406).

Better to understand the effect such a cornea has in refracting rays of light, let us suppose a cone of waves falling upon a convex refracting surface

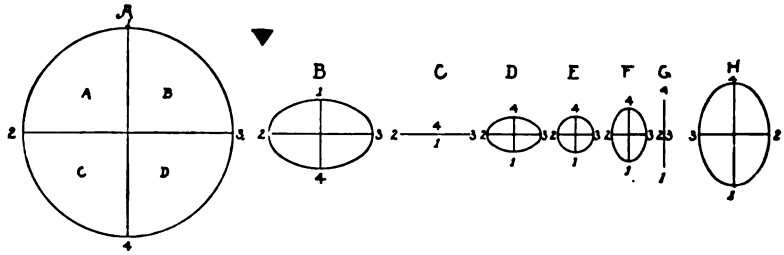


Fig. 407.—Refraction of rays in regular astigmatism.

with the shortest radius of curvature in the vertical, and the longest in the horizontal meridian. Through a central round opening (Fig. 407, *A*, *1*, *2*, *3*, *4*) let a cone of rays fall upon this surface. On account of the greater curve in the vertical direction *(1-4)* the rays will be turned up and down more than they will be turned in from the sides: they will converge faster vertically than horizontally, until at *B* they first form a horizontal oval, and then a horizontal band of light *(C)*, called the *first*, or anterior, *focal line*. The line is equal in width to the unfocused horizontal rays. The vertical rays, crossing, diverge until at *E* their height equals the width of the separation of the horizontal rays, thus giving rise to a circle. The vertical rays still becoming progressively wider and the horizontal rays narrower, the image circle *(E)* is converted into a vertical oval as at *F*, until at *G*, where, the rays in the horizontal meridian having been brought together, the vertical oval image is changed into a vertical line—the *second*, or posterior, *focal line*. The horizontal rays undergoing their crossing and the vertical rays becoming still more widely separated give width to an image at *H*, which becomes a vertical oval in shape or an ellipse with its long axis vertical. The interval *(C-G)* between these focal lines is called

Sturm's focal interval. The inequality of meridians producing regular astigmatism may take place either in a functionally emmetropic eye or in an hypermetropic or in a myopic one.

When one meridian is emmetropic and the other is hypermetropic, the condition is called simple hypermetropic astigmatism—Ah (Fig. 408). In this form the focus of the one meridian, usually the vertical, is on the retina, and the other, the horizontal one, is behind the retina.

When one meridian is emmetropic and the other is myopic, the condition is known as simple myopic astigmatism—Am (Fig. 409). The focus

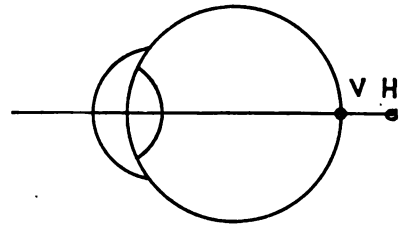


Fig. 408.—Simple hypermetropic astigmatism.

of the horizontal meridian is on the retina, and the focus of the vertical meridian is in front of the retina.

When both of the principal meridians are hypermetropic, differing only in degree, it is termed compound hypermetropic astigmatism—H + Ah (Fig. 410). The focuses of both principal meridians are situated back of the retina, that of the vertical being closest.

When both principal meridians are myopic, differing only in degree, it is known as compound myopic astigmatism—M + Am (Fig. 411). The

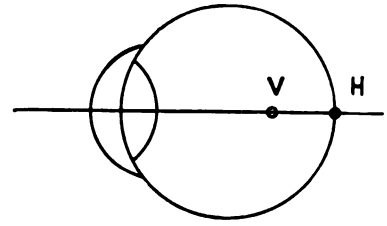


Fig. 409.—Simple myopic astigmatism.

focuses of both principal meridians are situated in front of the retina, that of the horizontal being closest.

When one meridian is myopic and the other is hypermetropic, the condition is called mixed astigmatism—Amh or Ahm (Fig. 412). The focus of the horizontal meridian is behind the retina, while that of the vertical lies in front of the retina.

A difference of one millimetre between the meridians of greatest and least curvature of the cornea produces an astigmatism of six dioptres.

Etiology.—Astigmatism is generally congenital and often is hereditary. It may also be acquired. The congenital astigmatism is principally regular

and dependent upon asymmetry of the cornea. In the majority of cases it is present in both eyes, though it may vary in degree. Donders has found that abnormal astigmatism occurs far more frequently in hypermetropic eyes than in others.

Acquired astigmatism is principally caused by inflammatory changes in the cornea, which lead to consecutive flattening of the cornea, and leave behind them opacities and cicatrices; it may also be caused by irregularity in the apposition of the edges of the incision after the operation of extraction of cataract and following iridectomy. It may likewise be caused by dis-

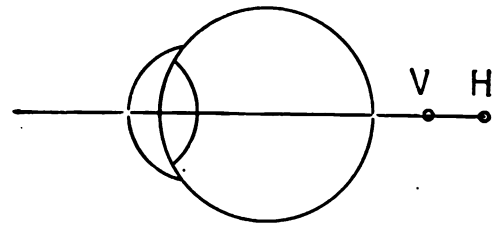


Fig. 410.—Compound hypermetropic astigmatism.

location of the crystalline lens, more particularly if it is obliquely displaced in the area of the pupil. It may be caused by swelling of the different sectors of the lens.

Symptoms.—SUBJECTIVE SYMPTOMS.—The patient will complain of a sense of weariness and sleepiness and of pains in the eye, in the temple, and in the forehead, which at times shoot into the back of the head. As astigmatism is one of the most frequent causes of disturbance of the extra-ocular muscle-balance, there may be present great discomfort when looking

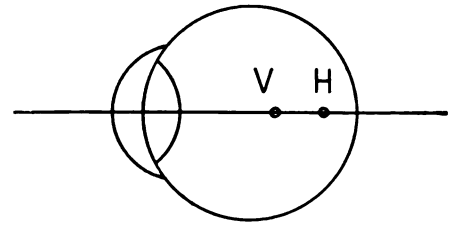


Fig. 411.—Compound myopic astigmatism.

at moving objects, vertigo, and difficulty in successively directing the gaze from one to another of different near points whose distances vary. The objects, before being fixed, appear indistinct or double. The patient is conscious of his eyes.

In astigmatism constant distinct vision is impossible for either distant or near work, because of the overlapping of the images of the different points of an object causing a blur, or a wrong impression of its outline. So long as astigmatism does not essentially diminish the acuteness of vision it is called normal. It is abnormal so soon as disturbance occurs. If the

eye is hypermetropic, distant horizontal lines will be seen more sharply than the vertical ones, because, the eyeball being short, the retina is nearer the anterior focal line, which is horizontal; while in the passive myopic eye vertical lines will be seen more distinctly than the horizontal ones, because the retina is nearer the vertical posterior focal line (Fig. 407).

When, by strain of accommodation, the hypermetropic eye makes itself emmetropic, the vertical lines will be seen the most distinctly. A distant point of light will appear as a horizontal oval in hypermetropic eyes in a state of rest, and as a vertical oval in myopia and emmetropia. Of course, this is true only when the shortest curved meridian is the vertical. It is impossible for the astigmatic eye to see simultaneously the strokes of letters which are at right angles to one another with absolute sharpness. Such a patient will make the characteristic mistakes in reading the Snellen test-card, mistaking E for P, Z for E, B for R, etc. The letters will have a streaked appearance.

They will also assert that certain radiating lines in some one of the astigmatic charts are the most plainly seen. If a stenopeic slit be placed before the eye so that the opening shall be parallel with the line best seen

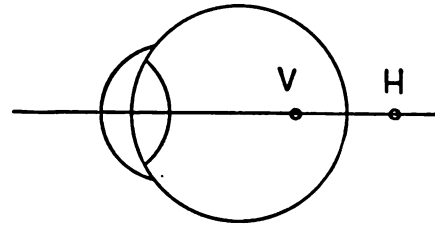


Fig. 412.—Mixed astigmatism.

on the dial-plate, and if attention be directed to the letters, the visual acuity will be found to be less with the slit so situated than it would be were the slit placed at right angles to this direction. The blurring of the image also greatly diminishes the range over which the patient can read small type. Persons suffering with astigmatism often assert that they are able to see better at certain periods of the day than at others, because the hour-hand and the minute-hand on the graduated face of a clock are seen better either when in a horizontal or vertical position.

Objective Symptoms.—The head is usually twisted to one side in accordance with the axes of the principal meridians of the astigmatism. In myopic astigmatism the eyelids are usually half-nipped in the effort to obtain good distance vision, whereas in hypermetropic astigmatism the eyelids are widely separated.

Ophthalmoscopic Appearance.—In astigmatism the optic disc will appear more or less oval instead of round. In direct ophthalmoscopic examination the enlargment is greatest in the meridian of greatest refraction, but the actual effect produced depends less upon the difference in the apparent length of the meridians of the disc than upon the contrast between

the sharp definition of all lines running parallel with one principal meridian and the indistinctness of those at right angles to this (Fig. 1, Plate IV).

With the indirect method, the contrast between the vessels is less marked, but the oval shape of the disc is readily seen. When the object-lens is held close to the eye, the longest diameter corresponds to the meridian of least refraction; as the lens is withdrawn from the eye, the meridian of greatest refraction elongates, either actually or relatively to the other meridian, so that the disc becomes circular and then elongated in the other direction. In high myopia the nerve was described as presenting an oval appearance due to its being viewed obliquely.

Diagnosis.—The presence of astigmatism may be determined by the characteristic mistakes in reading Snellen's type or by the appearance of the astigmatic chart. There are a great many different forms of these charts, but the one most used is the ordinary "clock-dial" (Fig. 413), which con-

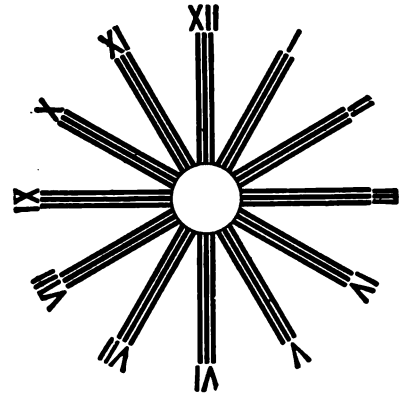


Fig. 413.

sists of a white card with peripheral Roman characters corresponding to the characters on the face of a clock. From those figures a series of three parallel black lines, with interspaces of the same width as the lines, cross from XII to VI, III to IX, etc. The card is placed on a horizontal line perpendicular to the patient's eyes, at the usual distance, one eye being tried at a time. The patient is requested to state which is the clearest, the blackest, and the most distinct line. The meridian of the eye which corresponds to the dark lines selected is the meridian of astigmatism. The axis of the cylinder will be opposite to the meridian of the dark lines. When the astigmatism is of low degree it may not be recognized by this means until the spheric correction is placed before the eye or after a cycloplegic has been instilled.

The presence of astigmatism may also be diagnosed by means of the stenopeic slit, Placido's disc, the perforated disc, Pray's letters, chromo-aberration test, the ophthalmometer, ophthalmoscope, retinoscope, or by the use of cylindric lenses. If the acuity of vision is diminished and can-

not be improved by spheric lenses and there is no organic disease, it usually signifies the presence of astigmatism.

IRREGULAR ASTIGMATISM is determined by the lowered acuity of vision; monocular diplopia and polyopia; the apparent deformity of objects looked at; and by the ophthalmoscopic appearance of the fundus oculi, the details of which appear elongated, compressed, or stretched, changing in shape as it is viewed through different parts of the cornea. If caused by conic cornea, the ophthalmoscope will show a circular shadow surrounding the central part of the red from the fundus. The keratoscope may also be used. Images reflected from the cornea are irregular and distorted.

Treatment.—Astigmatism should be corrected whenever it produces symptoms, no matter how small the amount of error. In the weak, sickly, and neurasthenic, asthenopia is produced by degrees of astigmatism which would pass unnoticed in the healthy and vigorous individual.

As to the ordering of glasses for the different forms of astigmatism, the same may be said as in the other refractive errors: there can be no fixed rule to follow blindly. The examiner must exercise his judgment. But in order to have some guide we may say that in mixed astigmatism and myopic astigmatism the full correction is ordered for distance. In myopic astigmatism the spheric lens may be made weaker for near work.

In correcting compound hypermetropic astigmatism the cylinder should be ordered full, combined with as much of the sphere, correcting the hypermetropia, as is necessary to secure normal muscle-balance for both distance and near. In many cases, especially if the astigmatism be of high degree, or if the axes of the cylindric lenses are oblique and asymmetrical, the distortion of objects produced by the correction of the error is so annoying to the patient that it may be necessary to give only a partial correction, and gradually increase the strength of the lens until the full amount that was found during the cycloplegic examination has been reached. At times, especially in old subjects, in order to make the patient comfortable it may be necessary to omit the cylindric corrections.

As a rule, in making the post-cycloplegic test in hypermetropic astigmatism the full correction should be placed before the eye, the strength and axis of the cylindric lens not being changed, but the spheric lens may be weakened until full acuity of vision is obtained, as in the treatment of hypermetropia. In a few rare cases the post-cycloplegic axis must be the permanent one that is used. In order to give good vision and restore muscle-balance it may be necessary to give one correction for distance and another for near work.

IRREGULAR ASTIGMATISM requires treatment only when the vision is very much reduced as the result of irregularities of the corneal surface from ulcers and cicatrices. It may be possible to find one meridian of regular curvature and by means of a cylindric lens vision may be improved. Vision is sometimes improved by stenopeic spectacles. An iridectomy may be useful by displacing the pupil toward a more regular portion of the cornea.

APHAKIA.

Definition.—By aphakia we mean an absence of the crystalline lens from the pupillary area. If the lens has by luxation or depression entirely disappeared from the plane of the pupil, even though it be still present in the eye, the condition is termed aphakia. Partial luxation, causing the equator of the lens to correspond to the plane of the pupil, is not aphakia.

When the crystalline lens is absent the power of accommodation is abolished. The lens, when in normal position, possesses an optical strength of about 11 D. Absence of the lens, therefore, in an eye previously emmetropic would create an hypermetropia of 11 D. The only eyes that do not become hypermetropic upon losing the lens are those which are myopic 11 D. or more.

It is claimed by some that in the lensless eye there is still a part of accommodation which is dependent upon the action of the ciliary muscle in diminishing the radius of curvature of the cornea. The apparent accommodation found in some cases after cataract operation is, however, probably due either to a slight shifting of the correcting lens, causing an increase in the distance between the glass and the eye, or to a narrowing of the pupil in the effort to see near objects, with consequent diminution of the circles of diffusion.

Etiology.—Aphakia may be congenital, but it is most frequently produced as a consequence of the resorption of the lens, either accidental or as the result of operation, and also as the result of the operation of extraction of a cataractous lens.

Symptoms.—The aphakic eye can see clearly only at the distance for which it is adapted by the length of its axis and the curvature of its cornea, with or without the aid of spectacles. The acuteness of vision is usually imperfect in aphakia following cataract extraction, due to turbidity of the surface of the pupil from a slight deposit on the inner surface of the capsule of the lens. There is generally an increase in the depth of the anterior chamber. The iris, deprived of its support, no longer bulges forward, as in the healthy eye. There is a certain degree of trembling (iridodonesis) with each sudden movement of the eye, unless the iris be rigid or adherent to the remains of the lens or its capsule. But none of these phenomena is characteristic.

The most decisive symptom of aphakia is the absence of the crystalline reflexes of Purkinje's images. In addition, in the normal eye the sectors and the direction of the fibres of the crystalline lens are easily seen on lateral illumination with light concentrated by a lens, particularly with the aid of a strong magnifying glass. Where these are wanting we may infer absence of the lens. Finally, a very high degree of hypermetropia, and a considerably weaker refraction in one eye than in the other, in connection with the form of the eye, render probable the existence of aphakia.

Diagnosis.—Absence of the lens is to be diagnosed accurately only by observing that the two images of Purkinje reflected from the lens are not present, and also by the inability to see the sectors and direction of the fibres of the lens on lateral illumination. It may also be suspected by the deep anterior chamber, trembling of the iris with sudden movement of the eye, and when after a blow or knock upon the eye the acuity of vision has suddenly diminished, associated with a very much higher hypermetropia than the fellow-eye without very manifest disturbances in the organ, and by absence of accommodation.

Treatment.—Aphakia following removal of the crystalline lens causes an hypermetropia of about 11 D. in the emmetropic eye. If the eye has been previously myopic, the hypermetropia will be reduced. The hypermetropia thus produced can be usually corrected for distance vision by a + 10 D. glass. A glass having a focus of 16 to 25 centimetres added to the above will be required for near work. Astigmatism should be sought for and should be corrected.

PRESBYOPIA.

Definition.—Presbyopia is the condition of any eye in which, as the result of age, the range of accommodation is diminished and the punctum proximum is removed beyond twenty-two centimetres (eight inches).

Etiology.—At birth the lens is soft, elastic, and perfectly transparent, and so nearly of the same index of refraction as the aqueous humor that in children it is often difficult and sometimes is impossible to demonstrate its presence by means of oblique light. It rapidly becomes more dense, especially the nucleus and the parts immediately surrounding it, until, after twenty years of age, it acquires a faint straw-yellow color, even though the lens is transparent, until it has become almost of an amber color, the nucleus increasing at the expense of the cortical substance. The result of this is a diminution in the elasticity; so that, even in early youth, the lens is less capable of swelling and changing its curvature than it was in infancy. However energetic the contraction of the ciliary muscle may be, and however complete the relaxation of the zonula of Zinn, the form of the lens varies less and less under their influence; and, with its convexity, its refraction, during the extreme effort of accommodation, differs less and less from that which it possesses when the eyes are at rest. In other words, the range of accommodation diminishes as age advances. Associated with the progressive increase in the rigidity of the lens there is late in life weakness or even atrophy of the ciliary muscle, which is an important factor in the causation of presbyopia.

The range of accommodation diminishes scarcely, if at all, more rapidly from much close work than it does in agriculturists, sailors, and others who, for the most part, look at distant objects. The same is true of the frequent use of the microscope or a magnifying glass as is required in the work of engravers and watchmakers; the regular course of the range of

accommodation is maintained despite much or little tension. In eyes predisposed to myopia much near work easily renders them more myopic, but it has no influence on the range of accommodation. There are morbid conditions which cause the range of accommodation, and sometimes also the amount of refraction, to diminish more rapidly than usual: general debility, the result of exhausting diseases, and premature old age.

If a person has quickly and repeatedly to strengthen his glasses, we should suspect the presence of glaucoma. The commencement of cataract also appears to hasten presbyopia, probably through more rapid hardening of the crystalline lens interfering with its mutability of form. Paresis and paralysis of accommodation interferes prematurely with the vision of near objects. Any disease that interferes with the nutrition of the lens will eventually limit its power to become more convex during accommodative effort. Also any disease which weakens the ciliary muscle hastens the advent of presbyopia.

Symptoms.—The actual fall of the accommodative power with the age and the range of accommodation for each period of life is very well demonstrated by Donders's diagram (Fig. 414). The figures on the left give the respective distances for which the eye can be accommodated, those below infinity being so marked as to express the distance at which the convergent rays, for which the eye is adapted in old age, would come to a focus behind its nodal point. The black curved line indicates the actual position of the near point at each time of life, as specified in numbers at the top of the table. The vertical lines joining the near and the far points give the entire range of accommodation. On the left-hand side are the equivalents in dioptres.

It is for emmetropia that this scheme was originally drawn. Hence the line commences at the zero-point of the division. It is straight and coincident with the zero-line until just before reaching the vertical line corresponding with the age 55 years, where it commences to descend and enters the negative portion of the diagram. The position of the punctum remotum does not change until the age of 55 years, when it changes its position and passes from infinity to the rear of the eye. The emmetrope commences to get hypermetropic, the myope notices a decrease in the myopia proportionate with the recession of his near point, and the hypermetrope is conscious of an annoying increase in his hypermetropia. Emmetropia and ametropia, without regard to the degree of the latter, are alike subject to the laws governing the range of accommodation.

If we consider presbyopia as commencing when the near point is removed beyond 22 centimetres, which is equal to 4.5 dioptres of positive refraction, then it will be seen by referring to Donders's diagram (Fig. 411) that a myope of over 7 dioptres can never become presbyopic, for even without accommodation they see at 142 millimetres up to the age of 50 years, and up to the age of 64 by adding to it their dynamic refraction. Even at the age of 80, when there is no longer any dynamic refraction and the static

has diminished 2.5 dioptres, they still have remaining $7 - 2.5 = 4.5$ dioptres with which to see at the required distance of 22 centimetres (8 inches). Myopes of lower degree than 7 dioptres become presbyopic, but earlier or later, according to the degree of their myopia.

The emmetrope becomes presbyopic at 40 years of age. The diagram shows that at this age the punctum proximum is at 4.5 dioptres. From this

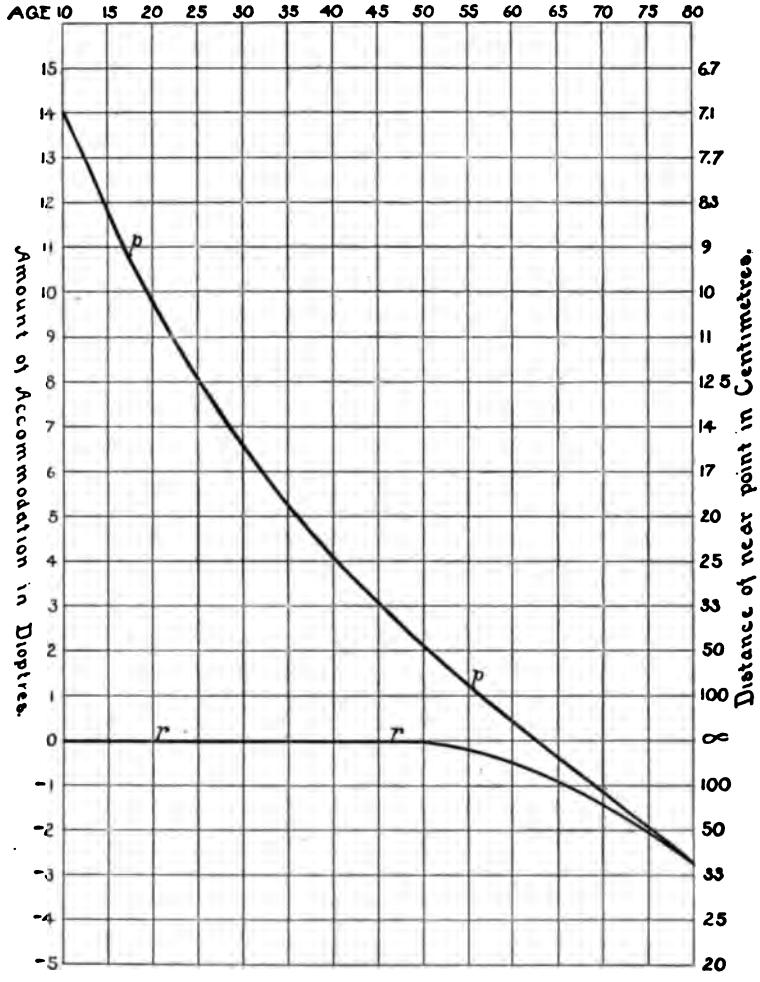


Fig. 414.—Range of accommodation at various ages. (DONDERS.)

time on the amount of positive refraction which the eye needs in order to have the requisite 4.5 dioptres must be supplied by convex lenses.

The hypermetrope becomes presbyopic sooner in proportion as his hypermetropia is of higher degree. Thus, a hypermetrope of 4 dioptres is presbyopic at the age of 25 years, because, in order to attain the required 4.5 dioptres of refraction, he must have $4 + 4.5 = 8.5$ dioptres of dynamic

refraction, and it is only up to the age of 25 that his accommodation is so strong as that (Fig. 414). When the hypermetropia is of higher degree, presbyopia manifests itself still earlier.

Thus it is seen that if we were to restrict the term presbyopia to the position of the uncorrected near point, the meaning of the word would be contradictory, for, as has been shown, the higher the degree of hypermetropia, the earlier in life would the eye become presbyopic, which term must lead to considerable confusion. But the hypermetrope is presbyopic only so soon as, in the use of glasses which neutralize the hypermetropia, the near point lies farther from the eye than 22 centimetres (8 inches).

The following table shows the near point and amplitude of accommodation in dioptres from 10 to 75 years, with 5-year intervals:—

YEAR.	NEAR POINT.	DIOPTRES.	YEAR.	NEAR POINT.	DIOPTRES.
10.....	7 cm.	14	45.....	28 cm.	3.5
15.....	8.5 "	12	50.....	40 "	2.5
20.....	10 "	10	55.....	55 "	1.75
25.....	12 "	8.5	60.....	100 "	1
30.....	14 "	7	65.....	133 "	0.75
35.....	18 "	5.5	70.....	400 "	0.25
40.....	22 "	4.5	75.....	....	....

At first, of course, no inconvenience is experienced from this gradual recession of the near point; we do not, in fact, notice it until the distance is so considerable that we cannot easily distinguish small objects. Seldom do we hear that close reading and work cause fatigue. The complaint is rather that vision is not accurate; the letters are not easily distinguished; numbers are confounded; a stroke is seen double. If we place small print in the hand of such a presbyopic person, he begins by holding the book too close to his eye, and does not distinguish; he subsequently very pathognomonically moves the book forward and the head backward, seeks a bright light, complaining of even ordinary print. Often, he may either hold the letters between the light and himself or so place them that a strong light shall fall both on the eye and on the printed page, not so much because the retinal images are by it more strongly illuminated, but because the pupil contracts; the circles of diffusion thereby become smaller and the retinal images less diffused. Therefore, also, the individual first perceives some difficulty in twilight, unless it be particularly strong. Inconvenience would have arisen even earlier, if the diminution of accommodation had not been accompanied with diminution of the diameter of the pupil. Thus, also, the small pupil of the old man makes the loss of the power of accommodation lighter to him: to this he is indebted for the fact that, even for distances for which he is not accurately accommodated, he still distinguishes tolerably well.

In full daylight in the open air a person can often, even in advanced presbyopia, read ordinary type, and this always succeeds on looking through a small opening.

Diagnosis.—Presbyopia is indicated by the age of the patient; the position of the near point; history of good distance vision and inability to see near small objects distinctly, being obliged to remove them farther from the eye, or even to seek a bright light, and to hold reading matter at an uncomfortable distance.

Treatment.—Having determined from the tests given that the symptoms complained of by the patient are due to a weakening accommodative power, which prevents the patient using his eyes with comfort for near work at a comfortable distance, this deficiency must be made up by substituting a convex glass of sufficient strength which will enable him to use his eyes with comfort and safety at the necessary distance.

As a rule, in the emmetropic eye the glass necessary to enable the patient to work comfortably at thirty-three centimetres (thirteen inches) will be a + 1 D. spheric lens at the age of 45 years, a + 2 sphere at 50 years of age, a + 2.50 sphere at 55 years, and a + 3 sphere at 60 years or over. This rule, as has been said, is applicable only to the emmetropic eye. Any existing ametropia must first be very carefully sought after and corrected, to which correction the above is added. For example, if there is an hypermetropia of 2 D. at 45 years of age, there should be a + 1 sphere added, which would make the correction a + 3 D. sphere.

All presbyopic hypermetropes should have a glass which represents the sum of the presbyopia and the hypermetropia.

In myopia the amount may be considered the equivalent to a convex glass for the correction of presbyopia; consequently a patient having a myopia of 1 D. would not require any glass for near work until he had reached the age of 50 years, when he would require a + 1 sphere. A myope of 3 or 4 D. never becomes presbyopic, inasmuch as he will always be able to read at his far point. In higher degrees of myopia it will be necessary to weaken the full correction until the patient can see near objects clearly. It will be found impossible to force the patient to read at thirty-three centimetres, because of the reduction of acuity of vision, which compels him to hold print closer to the eye to enable him to see clearly. Frequently high myopes will read more comfortably without any glass, holding the print close and using only one eye at a time.

In simple myopic astigmatism a convex cylinder of a strength equal to the concave cylinder with its axis reversed will enable the patient to read. For instance, if he requires a — 1 cyl. (axis, 180°) for distance, a + 1 cyl. (axis, 90°) will be required for near vision at 45 years of age. At 50 years of age a + 1 sph. ⊃ + 1 cyl. (axis, 90°), and at 60 years of age a + 2 sph. ⊃ + 1 cyl. (axis, 90°) will be necessary.

Compound myopic astigmatism amounting to several dioptres will require a reduction of the spheres only, leaving the cylinder unchanged. In lower degrees of compound myopic astigmatism the combination would be different; for instance, if distance vision required — 0.50 sph. ⊃ — 1 cyl. (axis, 180°) at 45 years of age, he would require — 0.50 sph. ⊃ + 1 cyl.

(axis, 90°); at 50 years his requirement would be + 0.50 sph. ⊃ + 1 cyl. (axis, 90°).

In mixed astigmatism the combination — 1 sph. ⊃ + 2 cyl. (axis, 90°) for distance will require at 45 years of age + 2 cyl. (axis, 90°); at 50 years, + 1 sph. ⊃ + 2 cyl. (axis, 90°).

It is best to place the distance and near correction in separate frames so that the optical centres of the glasses can be made to correspond to the visual lines when looking at a distance and when using the eyes for near work; otherwise there would be considerable prismatic deviation and distortion of objects produced by the cylindric lenses.

The rule of adding 1, 2, or 3 dioptres to the ametropic correction according to the age of the patient will answer when the patient wishes to use his eyes at thirty-three centimetres, but, as in the case of bookkeepers, musicians, carpenters, and those persons following any occupation in which it will require a longer range, as well as in those in which the object must be held closer, as with the engraver, goldsmith, and embroiderer, the distance at which the work is placed must be ascertained, and a glass should be given whose focal length will be somewhat greater than the distance required.

Great care must be taken not to give too strong a glass for the correction of presbyopia. A strong glass enables the eye to see distinctly without accommodative effort, but at a distance which makes considerable demand on the convergence. It is better to use the weakest glass that gives the required vision and have the optical centres set a little closer together than the centres of the pupils, the lenses thus acting as weak prisms with their bases inward, thereby diminishing the convergence necessary for binocular fixation, while they lessen the strain on the accommodation and bring the near point closer.

ANTIMETROPIA AND ANISOMETROPIA.

Definition.—*Antimetropia* means a different kind of refraction in the two eyes. It does not have reference to the amount of the error of refraction. One eye may be emmetropic, hypermetropic, or myopic (hypometropic), while the other eye is hypermetropic, myopic (hypometropic), or emmetropic.

Anisometropia means an unequal amount or degree of the same kind of refraction error in the two eyes. According to this definition, both eyes must be hypermetropic, myopic (hypometropic), or astigmatic, but one is more so than the other.

Etiology.—Antimetropia or anisometropia may be congenital or acquired. The difference in the development of the two eyes is usually associated with a similar inequality between the corresponding orbits and the two halves of the head. At the side where the strongest refraction occurs the orbit is situated closer to the median line, and its surrounding edges are placed more forward. But the connection is not absolute, for

the orbits may differ in form and position, while the two eyes may be emmetropic. It may even be that the less refracting eye corresponds to the more prominent side of the forehead, the more projecting cheek-bone, and the larger side of the chin. Acquired antimetropia is limited chiefly to aphakia and to loss of accommodation in one eye.

It is hardly possible that one eye may become myopic in consequence of its exclusive employment for near vision, while the other remains hypermetropic, for the organ which does not participate in vision partakes of the fatigue of its mate, and an eye excluded from vision and deviated may become more staphylomatous and more myopic than its fellow which is constantly active. The congenital predisposition to myopia is lacking in the active eye which has remained hypermetropic, while the other has become myopic.

Vision in antimetropia or anisometropia may be possible in three ways:—

1. Binocular vision. An antimetrope or anisometrope almost always makes the same effort of accommodation on both sides. He combines the distinct retinal image of the stronger eye with the diffuse image of the other. The difference between the two retinal images may be great (— 6 D. or more) without their ceasing to be united in one stereoscopic impression. But we usually abstract from the one eye more easily than from the other.

2. Vision with each of the two eyes alternately. This occurs especially when one of them is emmetropic and the other is moderately myopic, and when each possesses a good visual acuteness. The former is used in distant vision and the other for seeing near at hand.

3. Constant exclusion of the one eye, and always the same eye. This is especially the case when not alone the refraction, but also the visual acuteness, of the two eyes is very different. There is then usually divergence if myopic, or convergence if hypermetropic.

In the deviation inward the field of vision is diminished in the deviated eye and falls more over the other. This may produce confusion; therefore we mentally neglect the impressions received in the deviated eye, which consequently becomes amblyopic. In the deviation outward only a small portion of the field of vision is common. Therefore the power of vision is generally tolerably satisfactorily maintained, even though the eye is not used.

Diagnosis.—The presence of antimetropia or of anisometropia may be determined by the facial asymmetry, by the difference of the position of the near and far point of the two eyes, and by the same tests as are applied to errors of refraction previously mentioned.

Treatment.—In antimetropia with binocular vision, if one of the eyes is emmetropic and the other is ametropic, glasses should not be given for distance vision unless the ametropic eye has the better vision. For near work the patient should be given for both eyes the glass required by the

emmetropic eye. Antimetropia is never corrected by unequal action of the ciliary muscle.

If both eyes are ametropic and the difference is not greater than 1.50 D., with good vision in both eyes, we may equalize the refraction by giving the glasses which correct each eye. If the ametropia is greater than 1.50 D., we find which is the working eye and correct the ametropia, the same amount being given to the other eye.

If there is antimetropia without binocular vision, one eye being myopic 4 D. and the other emmetropic, the patient will use the emmetropic eye for distance and the myopic eye for near work, never having to wear glasses.

If both eyes are myopic we should give each the correcting glass of the weaker eye.

If both eyes are hypermetropic, and the more hypermetropic one is used for distance vision, each eye is given the correcting glass of the strongest hypermetropia. If there is an hypermetropia of 4 D. in one eye and 1 D. in the other, we may order + 4 D. for each eye. The first one will be used for distance and the other for near vision.

In antimetropia in which one eye is used exclusively it is only necessary to correct this one. The other should receive some form of exercise after having its refractive error corrected so as to retain some vision in case of accident to the good eye.

The treatment for antimetropia due to paralysis of accommodation will be found elsewhere in this book.

THE TRIAL-CASE is a box containing the lenses which are used in the examination of the refraction of the eye. These include convex and concave spheric and convex and concave cylindric lenses, which are in pairs; prisms, plane and colored glasses, opaque discs, and a stenopeic disc. A trial-frame and other accessories complete the equipment. The spheric lenses vary in strength from 0.12 D. to 20 D., and the cylindric lenses from 0.12 D. to 6 or 8 D. The interval between the lenses should be 0.12 D. for the lower strengths and 0.25 or 0.50 D. for the higher ones.

CHAPTER XXII.

THE OCULAR MANIFESTATIONS OF NERVOUS DISEASES.

By J. C. KNIPE, M.D., of Philadelphia,

Ophthalmologist to the Jewish Hospital.

VARIOUS external conditions of the eye are indicative of nervous diseases. Subjectively, there may be pain or anesthesia. Peripheral irritation of the sensory nerves will cause *neuralgias*, and paralysis of the same nerves will cause *anesthesia*. The latter condition of the cornea may be present in paralysis of the fifth nerve, which may later cause a vasomotor disturbance, and a *neuroparalytic keratitis* (Knies). Hysteric anesthesia of the cornea is not followed by keratitis.

Pressure on or injury to nerves, inflammation, or degeneration of the nerve-fibres or of the cells of their nuclei may cause *paralysis* of any of the ocular muscles. The lesion may be orbital or cranial, and may be due to meningitis, tumor, aneurism, fracture, hemorrhage, embolism, thrombosis, abscess, hydrocephalus, changes in the blood-pressure, edema, softening, and lithiasis. In *congenital paralysis* the muscle may be absent: *e.g.*, congenital ptosis from absence of the levator palpebræ superioris.

If all the eye-muscles (except the superior oblique and the external rectus), together with the levator palpebræ superioris and the iris and ciliary muscles, are paralyzed, the third *(motor oculi)* nerve is involved. The *superior oblique* and the *external rectus* depend, respectively, on the fourth and sixth nerves.

Conjugate lateral paralysis, in which the eyes fail to move conjointly to the right or left, is due to a lesion in the centre for associated movements, the situation of which has not been positively determined. An irritation of the cortical region of the fifth nerve will produce conjugate movements to the opposite side, and it has been shown that destructive lesions of the cortex will produce a *paralysis of associated movements* to the opposite side. The antagonists draw the eyes away from the paralyzed side of the body, but toward the side of the brain lesion. If the lesion is in the pons, the opposite side of the body is paralyzed, but by the involvement of the nucleus of the sixth nerve—which is in close proximity on the same side of the pons—the external rectus muscle of the same side is paralyzed, and the eye turns in the opposite direction—toward the paralyzed side of the body. Irritative lesions in the same situations would have the opposite effect.

Ptosis (paralysis of the levator palpebræ superioris) is usually part of the paralysis of the third nerve. It may exist independently by involvement of only a branch of that nerve. A lesion of the cortical trigeminal (fifth) nerve seems to produce paralysis of the opposite levator palpebræ superioris (Knies). It may also exist independently on the same side as

the lesion in disease of the pons, associated at times with conjugate paralysis. Ptosis may also exist independently in crossed paralysis with a lesion in the crus cerebri. Paralysis of a portion of the third nerve, particularly the branch to the levator palpebræ superioris, may be caused by a lesion of the cerebral peduncle (Swanzy).

Fourth-nerve paralysis is rarely found alone, and is usually produced by disease at the base of the brain, such as meningitis or pressure in the valve of Vieussens. (Pfungen.)

Paralysis of the sixth nerve is more frequent as a distant symptom than any other (Swanzy), particularly in lesions of the cerebellum (Wernicke). This is said by Gowers to be due to the lengthened course of the sixth nerve over the pons, making it more liable to pressure. Fracture at the apex of the petrosal part of the temporal bone may produce it.

Paralysis caused by *basal lesions* in the neighborhood of the pons, the ventricles, and the aqueduct of Sylvius can be distinguished from *nuclear paralysis* by the association in the former of graver symptoms such as hemiplegia, vomiting, headache, optic neuritis, and hemianopsia.

Irritative lesions, in contradistinction to destructive lesions, may cause *muscular spasm*. Spasmodic movements of the levator or of a rectus muscle are common. The orbiculares may be spasmodically contracted in hysteria, simulating double ptosis, but differentiated by a subjective muscle resistance which is not present in true paralytic ptosis. A temporary ptosis or diplopia is a symptom which will cause strong suspicion of tabes or syphilis.

Nystagmus is another spasmodic condition of the ocular muscles. It is characterized by rhythmic oscillations of the eyeballs. It is almost always present in insular sclerosis and Friedreich's hereditary ataxia, and may be a symptom of meningitis, cerebral hemorrhage, and tumors, or an hysteric manifestation.

CONDITIONS OF THE PUPIL.

Miosis.—Caused by an *irritation* of the pupil-contracting centre. This symptom is present in the early stages of inflammatory affections of the brain, in cerebrospinal and tubercular meningitis, and early in tumors and apoplexy. Berthold pointed out that, in this, apoplexy differed from embolus, in which the pupils are not affected. It is also noticed in the beginning of hysteric and epileptic attacks, in tobacco-amblyopia, and in watchmakers and other habitual near workers.

Miosis caused by *paralysis* of the cervical sympathetic or the pupil-dilating centre occurs in injury to the cord, pressure of an aneurism or of enlarged glands, and in lesions of the spinal cord, as in locomotor ataxia. It occurs also in general paralysis of the insane.

Mydriasis caused by an *irritation* of the pupil-dilating centre, is present early in inflammation, anemia, or tumor of the cervical cord. It may also occur in intestinal irritation, mental excitement, early in acute mania, and in general paralysis of the insane.

Mydriasis caused by *paralysis* of the pupil-contracting centre occurs

from pressure on the third nerve at the base of the brain or in the orbit. It also occurs in glaucoma, in the later stages of general paralysis of the insane, and in optic-nerve atrophy in which there is no light-stimulus.

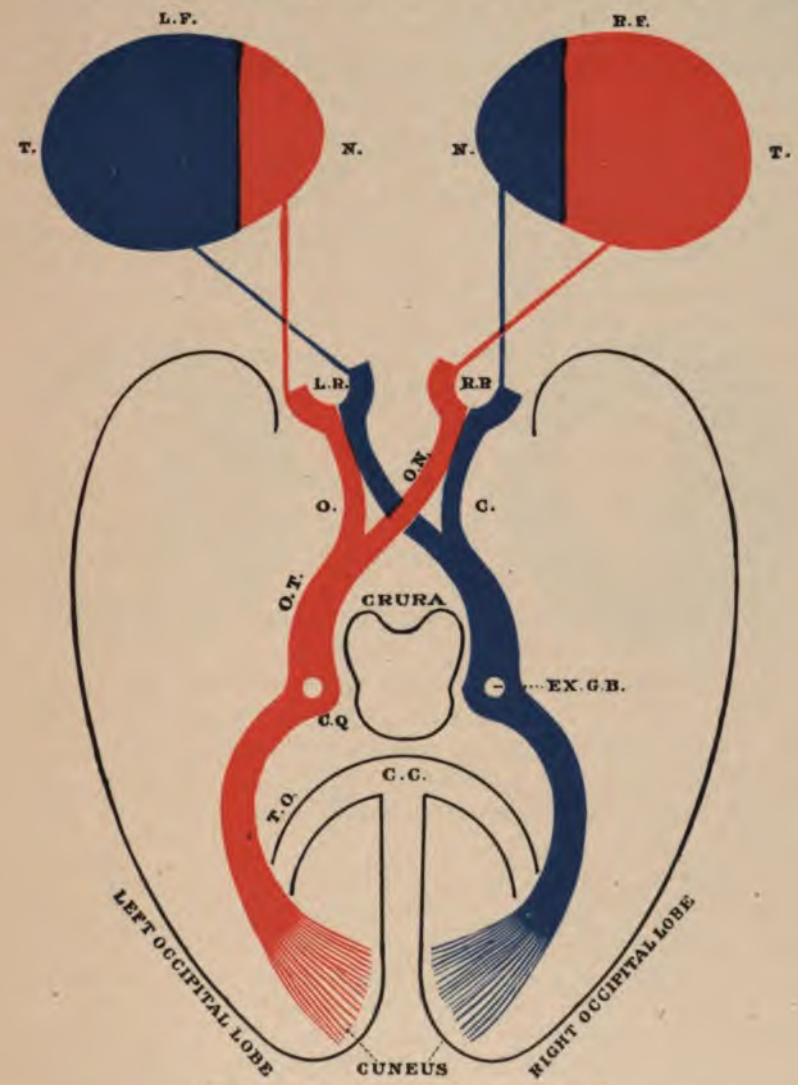


Fig. 415.—Diagram of the visual tract.

R. F., Right visual field. *L. F.*, Left visual field. *N.*, Nasal side. *T.*, Temporal side. *R. R.*, Right retina. *L. R.*, Left retina. *O. C.*, Optic chiasma. *O. N.*, Optic nerve. *O. T.*, Optic tract. *C. Q.*, Corpora quadrigemina. *EX. G. B.*, External geniculate body. *T. O.*, Thalamus opticus. *C. C.*, Corpus callosum.

THE VISUAL TRACT.

The visual tract (Fig. 415) begins with the rods and cones of the retina and the three bundles of optic-nerve fibres making up the papilla and the optic nerve. This extends back to the chiasma, in which the inner

bundle of each nerve decussates and joins the outer bundle of the opposite nerve and becomes the optic tract. The tract extends around the crus cerebri, and ends in the external and internal geniculate bodies, and the pulvinar on the posterior part of the optic thalamus. Some of the fibres extend to the corpora quadrigemina. These parts are called the primary optic centres, and from them the visual radiations, or fibres of Gratiolet, extend backward through the internal capsule to the cortex of the cuneus and the superior occipito-temporal convolution. Henschen believes from investigations that the centre of vision is in the middle of the calcarine fissure. Each hemisphere is in anatomic relation with the corresponding lateral half of each retina. At the macula lutea these halves seem to overlap, making a double nerve-supply, by which means central vision is preserved in cases of a destructive lesion on one side producing hemianopsia. When, however, there is an aneurism of the central artery or vein, or an inflammation of the optic nerve, the fibres in close proximity supplying the macula are implicated, and central blindness or a scotoma results. This is seen in tobacco- and alcohol- amblyopia.

THE FIELD OF VISION.

A lesion which obstructs the visual pathway beyond the chiasma or destroys the visual centres in the cuneus causes lateral homonymous hemianopsia.

Hemianopsia or Hemianopia (Half-blindness).—If the blind half be toward the same side in both eyes, it is called *homonymous;* if on opposite sides, it is known as *heteronymous hemianopsia.* It may be *complete* or *incomplete,* according to the involvement being of one-half the field or merely a sector of it. It may be *absolute* or *relative,* as the blind field is totally or partially blind. The hemianoptic field usually shows an indenture at the point of fixation corresponding to the overlapping of the nerve-supply to the macula.

The right optic tract contains all the nerve-fibres going to the right halves of both eyes. Its division would cause half-blindness on the opposite side of the field of vision. Those on the nasal side decussate to the right half of the left eye. Those on the temporal side continue forward on the same side (see Fig. 416).

Longitudinal division of the chiasma would cut off all the fibres to the nasal halves of the retinæ, and produce *double hemianopsia*—blindness to the right for the right eye and to the left for the left eye. Pressure on the third ventricle or enlargement of the pituitary gland may be the cause. A *lesion of the chiasma laterally* would cause blindness on the temporal side of the retina. In rare instances *bilateral blindness* in the nasal fields has been produced by corresponding lesions on each side affecting the non-decussating fibres. A *double temporal* or *nasal blindness* can occur only in diseases of the chiasma. The chiasma may be the seat of tumors or syphilitic growths or may be pressed upon through the third ventricle

in hydrocephalus. Gouty changes and interstitial hemorrhage have been observed. Migraine and hysteria may cause temporary hemianopsia. *Superior* and *inferior,* or *altitudinal,* hemianopsia is very rare. The optic tract may be involved by traumatism, hemorrhage, softening, basilar tumors, or thickening in multiple sclerosis. One eye may be entirely blind from degeneration of or from injury to the optic nerve, or from tumor, aneurism, basilar fracture, or caries of the sphenoid bone.

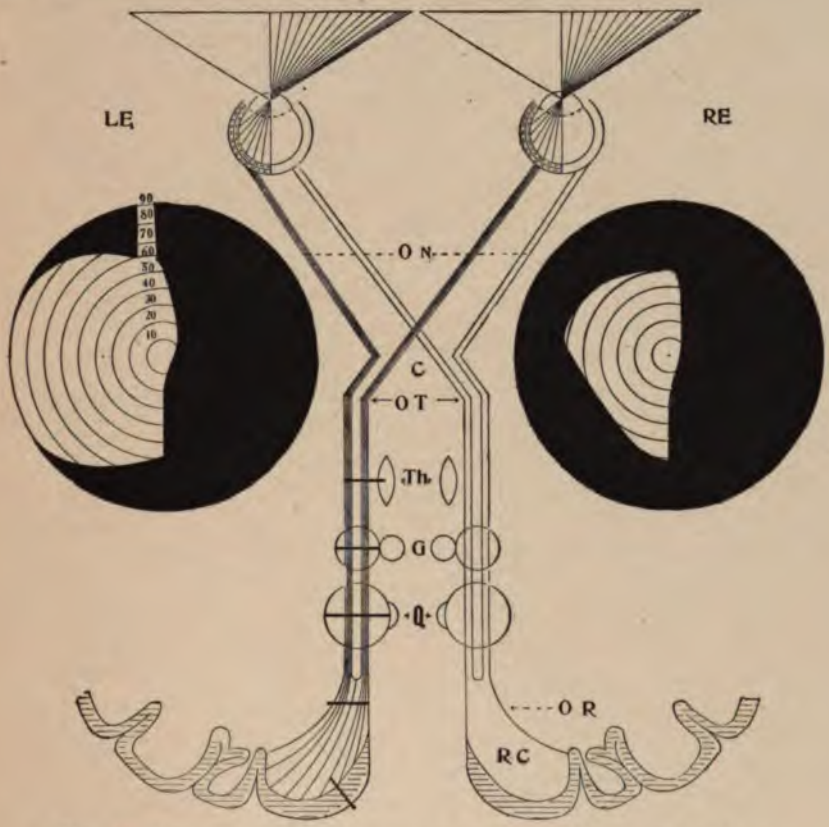


Fig. 416.—Diagram of right homonymous hemianopsia and of the sites of lesions which may cause it.

RE, Right eye. *LE*, Left eye. *ON*, Optic nerves. *C*, Chiasma. *OT*, Optic tracts. *Th*, Thalami optici. *G*, Corpora geniculata. *Q*, Corpora quadrigemina. *RC*, Right cuneus. *OR*, Optic radiations.

FOCAL LESIONS OF THE BRAIN.

Brain diseases are the most frequent causes of both optic neuritis and optic-nerve atrophy. The disease may be *focal* or *diffuse*. Focal lesions producing optic neuritis are usually caused by tumors, but foci of softening, abscesses, thrombosis of the sinuses, aneurisms, apoplexies, and cysts may be the pathologic factors. *Optic neuritis* may be the first and only evidence of the disease. Even the vision may not fail until post-neuritic atrophy sets

in. Recurrent *transient loss of vision* may be a prominent symptom of brain disease whether focal or diffuse. Similarly situated focal lesions may give rise to very different symptoms. There may be merely an *irritation* of the centre causing increased functional activity, or a *paralysis* of the centre may destroy the function of the part. If between the two, there is a *paresis* of the centre, with only a partial loss of function.

The most prominent symptom of focal disease may be the disturbance in the field of vision, usually *hemianopsia.* If the lesion exists anywhere

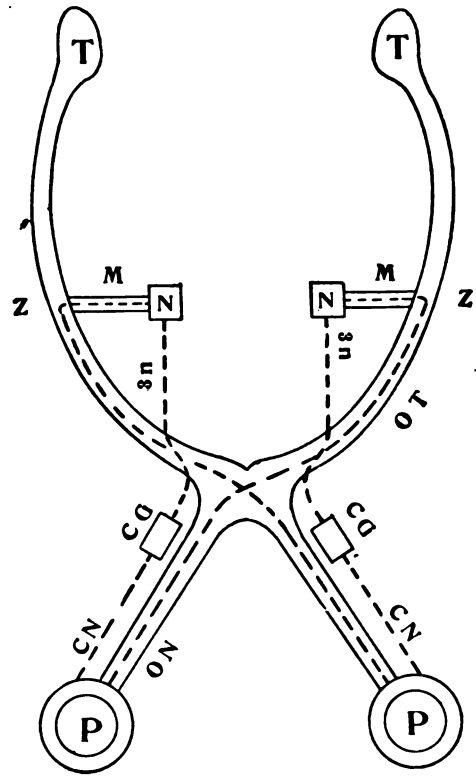


Fig. 417.—Diagram of Wernicke's pupil symptom.

P, The pupil. *T*, Centre for vision. *ON*, Optic nerve. *OT*, Optic tract. *M*, Meynert's fibres from *Z* (the geniculate body) to *N* (the nucleus of the third nerve). Thence by the third nerve to *CG* (the ciliary ganglion) and by the short ciliary nerve (*CN*) to the sphincter pupillæ (*P*). A lesion beyond *Z* would not disturb the pupillary reflex. *3n*, Third nerve.

between the cortical centre and the chiasma,—for example, in the right optic tract,—the nerve-fibres going to the right half of each retina will be cut off, and the left fields of vision will be cut out—*left homonymous hemianopsia.* If the lesion is in the chiasma, the decussating fibres to the nasal side of the retina will be involved, and the opposite fields may be blind—*bitemporal hemianopsia.* If the lateral fasciculi of both sides of the chiasma should be coincidently involved, the temporal halves of the retinæ would be cut off and *binasal hemianopsia* would result.

By means of the *hemianoptic pupil-symptom* of Wernicke a differential diagnosis between lesions of the tract and the cortical centre may be made. If the pupil responds to light thrown on the amaurotic retina, the lesion is back of the geniculate bodies. The presence of this reaction indicates that the pupillary nerves which accompany the optic tract as far as the geniculate bodies are not involved. There the afferent pupillary nerve leaves the tract by way of Meynert's fibres to become the efferent impulse to the pupil, going first to the gray matter of the aqueduct of Sylvius, then to the third-nerve nucleus, and from there through the short root of the ciliary ganglion to the ciliary ganglion and the short ciliary nerves to the sphincter pupillæ. If the lesion is in the path of the pupillary reflex, the pupil would fail to react (see Fig. 417).

Visual Amnesia, or Mind-blindness, is another symptom which may be present in focal brain trouble. The patient perceives an object perfectly, but has no memory of what it is. By closing the eyes it may be recognized by touch or by the other senses. This symptom is present when the lesion is in the centre for visual memory or in the path connecting the centre for vision with the centre for visual memory. In these higher visual centres both eyes are represented in each hemisphere. They are situated in the angular gyri of the parietal lobes, and communicate with each other through the callosal crossway. A lesion in this region does not cause hemianopsia.

Alexia, or Word-blindness, consists in the inability to read written or printed words, although the same words may be used fluently in conversation. Individual letters can be usually recognized *(subcortical alexia)*. It may be combined with an inability to write *(agraphia, cortical alexia)*. According to Dejerine and Wernicke, word-blindness with agraphia is due to a lesion in the left angular gyrus and inferior parietal lobule—the centres for visual word-memories.

Visual Aphasia consists in inability to name an object which the patient sees and of which he knows the use. He always gives the object a wrong name. If the eyes are closed, the other senses will recognize the object. Freund places the lesion in the left occipital lobe in the path from the centre of visual memory in each hemisphere to the speech-centre in the left hemisphere.

Dyslexia is a disgust for reading after a few words or lines are gone over. It may precede grave brain trouble. The seven or eight cases on record were all fatal. According to the autopsies, the lesion is around Broca's lobe.

Amnesic Color-blindness.—The patient cannot name the color of an object, although he distinguishes color and can perform color-tests perfectly.

Visual Hallucinations may occur in the blind fields in cases of hemianopsia which should not be mistaken for the visual hallucinations of insanity. They are supposed to be due to an irritation of the visual memory-centre.

OCULAR SYMPTOMS OF ORGANIC DISEASES OF THE BRAIN AND SPINAL CORD.

Meningitis.—1. Pachymeningitis, or Inflammation of the Dura, may cause *optic neuritis*. When the inflammation and pressure are at the base of the brain, the *fields of vision* may be affected from pressure on the optic tracts or nerves; and pressure on the third and sixth nerves may cause *ocular palsies*. Involvement of the fifth nerve may cause pain or insensibility in the regions which it supplies. Inflammation of the dura is less severe than is that of the pia mater.

2. Leptomeningitis (Acute Cerebral Meningitis; Cerebrospinal Meningitis).—There are early external inflammatory symptoms, such as conjunctivitis and swelling of the lids. The cornea may be infiltrated. The pupils at first are usually contracted, and later dilated, or they may be immovable. After a few days the optic disc may become hazy and the vessels are enlarged. The *papillitis* may be severe, the disc becoming obscured and subsequently atrophied. Blindness may ensue. Retinal hemorrhages and neuroretinitis are not infrequent. The severe infection may cause purulent iridochorioiditis and panophthalmitis. The third nerve is usually affected, and *strabismus*, or a loss of conjugate action, results.

3. Tubercular Meningitis.—Miliary tubercles are often present in the chorioid, but Cohnheim has pointed out that they are not as frequently observed as in general tuberculosis without meningitis. The base of the brain is usually involved in tubercular meningitis, in consequence of which *optic neuritis* and the *orbital paralyses* are more frequent than in other forms of meningitis.

Multiple Sclerosis (Disseminated Sclerosis) is rich in eye symptoms, upon which much depends for its early diagnosis. Any portion of the eye may be affected. The pupils are likely to be contracted, and unequal in size, with diminished reaction to light and accommodation, but the Argyll Robertson pupil is very rare. According to Uhthoff, the extrinsic eye-muscles are involved in 17 per cent. of the cases. The disturbance is usually bilateral and transient. One muscle only may be paralyzed or there may be complete *ophthalmoplegia*. The nuclear nature of the disease is sometimes shown in the lack of associated movements. *Nystagmus* is a very frequent symptom, true nystagmus being present in 20 per cent. of the cases, and the remainder usually show nystagmiform movements—the *ocular ataxia* of Swanzy. These are shown by directing the patient to look from the usual line of sight: *e.g.*, up and out.

Optic-nerve atrophy occurs in about 50 per cent. of the cases. The process is generally a slow one, and is seldom extreme. Vision may be more or less affected. A transient form of blindness is not uncommon. Hemianopsia is never present, which, as a localizing symptom, places the lesion, as far as visual symptoms are concerned, peripheral to the optic commissure, although the lesions causing the different ocular palsies may be as

far back as the nuclei of the nerve governing the muscle. The occasional disturbances in the field of vision are *central scotoma*, irregular and regular *retraction of the field*, and *dischromatopsia*, particularly for red and green.

Locomotor Ataxia (Tabes Dorsalis).—Probably no other nervous disease has so many characteristic eye-symptoms, many of which are present long before the ataxic symptoms show themselves, and, indeed, the diagnosis is often made from the early ocular symptoms.

There is apt to be a contraction of the pupil, in some cases almost to a pin-point size *(spinal miosis)*, although the pupil may react promptly to light and convergence. This condition is to be considered as either a paresis of the pupil-dilating fibres from disease in the front part of the aqueduct of Sylvius or as a paresis due to disease of the cilio-retinal centre in the lower part of the cervical cord. The pupils may be normal, or they may be accidentally dilated as a result of a general third-nerve paralysis. The *Argyll Robertson pupil* is found most frequently in this disease. In this the irides do not reach to light, or, if they do, it is but very sluggishly, but they react promptly in convergence. This symptom may be present when there is miosis, when the pupils are normal, or—as more generally occurs—when there is some inequality of the pupils *(anisocoria)*. This is an early symptom of tabes, and it may make the diagnosis reasonably certain long before the development of any ataxic gait.

Paresis of one or more of the ocular muscles with its accompanying diplopia occurs in 30 per cent. of the cases of tabes. These may be transient or permanent in nature. The sixth nerve seems to be the one most often attacked, but the third is not infrequently affected, together with its branch to the levator palpebræ, causing a true ptosis. Paralysis of accommodation occurs rarely in this disease, and is nearly always a late symptom.

There is often a narrowing of the palpebral fissure and a slight drooping of the upper lids, but, as the patient can by his will-power open them normally, it is not to be considered as a true ptosis. On closing one or both eyes there is often a marked twitching of one or both orbiculares. This symptom is not of much importance, as it is not infrequently present in persons who are perfectly well.

Epiphora is a symptom mentioned by some authorities, and is due probably both to a relaxation of the lower lid and to hypersecretion of the lacrimal gland. Berger claims to have frequently found a reduction of intra-ocular tension, but this contention is not borne out by other observers.

Some authorities mention *nystagmus* as a symptom of tabes, but probably Swanzy's term, *ocular ataxia*, is better. This consists in a nystagmus developing when the patient attempts to fix an object with his eyes, but which ceases the moment the eyes are at rest.

A symptom which occurs in at least 30 per cent. of cases is *optic-nerve atrophy*. It is an early rather than a late symptom, and cases are on record where it has preceded the ataxic gait twenty years. The atrophy is nearly

always bilateral, although generally more advanced in one eye than in the other. As the atrophy advances there is a *concentric contraction of the visual field,* especially so to the outer side. Central vision may remain good for a long time. Sometimes the field shows only a sector-like defect. As a result of the atrophy, color-blindness is not infrequent, the recognition of green being lost generally before that of red. The inequality of the pupils is probably explained by the atrophy of one optic nerve being much more advanced than that of its fellow.

Pseudobulbar Paralysis of Cerebral Origin.—This affection usually has no eye-symptoms. If they are found, they serve to establish a differential diagnosis from true bulbar paralysis, which has no eye-symptoms. When ocular symptoms are present they may consist of any one or more of the following: Optic neuritis, optic-nerve atrophy, and impossible or difficult voluntary closure of the eyelids, while reflex closure is normal. Other voluntary movements of the eyeball may be impossible, although all involuntary movements take place.

Combined Sclerosis of the Spinal Cord (Ataxia Paraplegia of Gowers; Postero-lateral Sclerosis).—Inequality of the pupils, irregularity of outline, and sluggishness in the mobility of the iris are frequently seen in this affection. The light-reflex is rarely lost.

Friedreich's Ataxia (Family Ataxia).—Hereditary ataxia bears considerable resemblance to locomotor ataxia, but its ocular manifestations are exceedingly limited as compared to the latter affection. Nystagmus and nystagmiform movements, increased by effort, are the most marked ocular symptoms. In Friedreich's form, optic-nerve atrophy is very rare. In Marie's form, the hereditary cerebellar ataxia, it is a common symptom, as is also a loss of pupillary reflex, which is not found in Friedreich's type. In Marie's type, changes in the form- and color- fields have been noted, together with a loss of visual acuity.

Acute Ascending Paralysis.—This is rarely accompanied by ocular symptoms, but there may be ocular palsies, paralysis of accommodation, and mydriasis.

Myelitis.—When optic neuritis is associated with myelitis of the cord, it is due to a similar process in the optic nerve. If the disease is in the lower cervical or the upper dorsal region, the cilio-retinal centre may be irritated and mydriasis results, or it may be paralyzed and cause miosis.

Multiple Neuritis.—Toxic agents—such as alcohol, lead, arsenic, and carbon gases—which may cause multiple neuritis may also produce a toxic amblyopia. The vision may be reduced. The pupils have been observed to be dilated, contracted, or irregular. The accommodation is usually disturbed in diphtheritic neuritis. In the latter disease diplopia occurs in 10 per cent. of cases, and less frequently in alcoholic neuritis. The motor nerves of the eye may be singly involved, as in ptosis, but there may be complete ophthalmoplegia, which is usually nuclear in origin. Optic neuritis, contracted fields, and color-scotoma may complete the eye-picture.

Migraine may cause portions of the eyebrow or the cilia of the lids to turn gray. The attacks may be preceded by scintillating scotomata or visual hallucinations. Optic-nerve atrophy sometimes sets in where the disease has been persistent for some time. It is likely to be mistaken for glaucoma. Forms of oculomotor paresis are not infrequent, and a special classification of "ophthalmoplegia migraine" has been suggested.

Neuralgia.—This occasionally gives rise to canites. Optic-nerve atrophy is sometimes found, and these cases should be suspected of glaucoma. Temporary amaurosis is not infrequent. Neuropathic keratitis with ulceration of the cornea may be due to interference with the trophic centre of the fifth nerve.

Herpes Zoster.—This occurs in the distribution of the fifth nerve, especially the ophthalmic branch. Blebs may form on the cornea and the sight may be more or less reduced. They heal with a disfiguring opacity.

General Paralysis of the Insane.—General paresis is rarely accompanied by optic-nerve atrophy. It is apt to occur early in the disease. The most important pupillary sign is the absence of light-reflex. Miosis may be extreme in the early stage, followed in the late stage by mydriasis. There is variable inequality of the pupils. Simple constant inequality is met with in ordinary insanity.

The *paradoxic pupil,* in which the pupil first contracts to a beam of light, then partly dilates, and then oscillates, and finally remains dilated widely regardless of the light, is a forerunner of paresis. There may be a loss of consensual pupillary reflex. The third and sixth nerves are sometimes involved early. Ptosis, transient nystagmus, and twitching of the lids may occur. Mind-blindness may come on in the late stages. The field of vision is not impaired.

Insanity.—Fundal and other ocular disturbances are so rare as to be considered coincident. Visual hallucinations may annoy the patient.

Paralysis Agitans.—A fine fibrillary tremor takes place along the margin of the upper lid, especially marked when the eye is closed.

Infantile Paralysis.—Usually no eye-symptoms are presented, but optic neuritis, hemianopsia, and paralysis of the ocular muscles have been noted.

Tetany.—A form of nuclear cataract which progresses very slowly is sometimes found.

Hydrocephalus.—The ocular symptoms of this disease are those which result from pressure on the optic chiasma by the distended third ventricle, namely: bitemporal hemianopsia, optic neuritis, and optic-nerve atrophy, the latter being secondary or primary according to the severity of the attack. Strabismus is not uncommon.

Acromegalia.—The eyebrows are coarse and heavy. Thickening of the bony walls of the orbit may cause exophthalmos and intra-ocular pains. Pressure from a tumor of the pituitary gland may cause double optic neuritis and optic-nerve atrophy. Ocular palsies and nystagmus may be present.

Thomsen's Disease (Myotonia Congenita).—This rare disease is characterized by a rigidity of the voluntary muscles. It is seen in different generations of neurotic families, and is either a disease of the muscles themselves or a trophoneurosis. There is at times a disorder of the muscular apparatus of the eyes. The lids may partake of the general muscular stiffness. Amblyopia of a temporary character has been observed.

Syringomyelia and Morvan's Disease are characterized by sensory and trophic disturbances, and cause, in some cases, concentric contraction of the field of vision without any fundus changes discernible.

Epilepsy has no characteristic eye-symptoms, although few, if any, attacks of epileptic convulsions are unaccompanied by more or less of them. The most frequent is *visual aura*—a subjective symptom elicited from the patient. He may describe it as a sensation of light, color, flames, or flushes. Visual aura is strongly indicative of organic trouble in the occipital lobe, especially if homonymous. *Conjugate lateral deviation* of the eyes to the opposite side of the body from that in which the convulsive movements began may occur at the beginning of an attack. The head inclines in the same direction. Later the eyes turn strongly in the opposite direction.

The action of the pupils during an attack is varied. They may be strongly contracted or widely dilated, or they may be both during the same attack. A rapidly changing pupil after an attack of unrecognized nature is diagnostic of epilepsy. The ophthalmoscopic appearance of the fundus during a convulsion may be normal, or there may be extreme pallor of the disc with contraction of the vessels, or hyperemia of the disc with dilation of the vessels. Following the convulsion there is often a transient concentric narrowing of the field of vision and lowered acuity of vision.

Subconjunctival ecchymosis and opacity of the lens or complete cataract following an epileptic seizure are to be regarded as accidents rather than symptoms of the disease.

We have been considering epilepsy and its ocular symptoms as of organic origin, but it should also be considered as a reflex phenomenon, due to an irritation, the seat of which may be in the eye itself. An error of refraction, especially one of high degree, or much astigmatism is sometimes the cause, and its correction may prevent or ameliorate the attacks. In the same manner the correction of muscular insufficiencies by advancements and tenotomies has effected cures, but the results have not been as brilliant as could be reasonably expected.

Chorea.—Blepharospasm is sometimes a forerunner of a general attack of chorea. As in epilepsy, errors of refraction seem at times to be the cause of chorea, and wearing the proper correction may cure the disease. In the few cases reported of embolism of the cerebral artery accompanying chorea the coexistence of endocarditis with which chorea seems to be so intimately connected has been assigned as the cause of the emboli. Chorea not infrequently shows a low-grade neuroretinitis, and even a decided papillitis manifests itself at times.

Exophthalmic Goitre (Graves's Disease; Basedow's Disease).—Various theories have been advanced as to the cause of exophthalmic goitre. They point to a nervous origin to account for the three pronounced symptoms, namely: *exophthalmos, enlargement of the thyroid,* and *tachycardia.* The last is the most constant. Either of the others may be wanting. Until recently a lesion of the cervical sympathetic was supposed to cause a paralysis of the vasomotor nerves producing vascular dilation of the carotids and the vessels supplying the thyroid and the orbits, the tachycardia resulting from irritation of the excitomotor nerves of the heart. Division of the sympathetic experimentally in animals has failed to produce these results. Filehne divided the restiform bodies and produced an exophthalmos, but no tumefaction of the thyroid. He believes, in accordance with Sattler's theory, that there is a lesion of an area of the brain the nerve-fibres of which pass through the restiform body, the vagus being involved to account for the tachycardia, the exophthalmos and goitre being caused by the absence of the vasomotor supply—and the consequent vascular dilation.

The eye-symptoms may first direct attention to the disease. The *exophthalmos,* or *proptosis,* may be the most prominent. It may be noticed that when the eyeball is directed downward the upper lid does not follow it steadily, but in a jerky manner *(von Graefe's sign).* The upper lid is more or less retracted *(Dalrymple's sign).* These two symptoms may be wanting. Owing to the proptosis, the lids do not come together during the act of winking, and this gives rise to *Stellwag's sign,* which is an imperfect and abnormal infrequency of nictitation. There may be a minute's time between the involuntary winking movements. The increased exposure of the cornea may permit it to become covered with pannus. During sleep the cornea may remain uncovered, and sloughing may follow from inability to close the lids. *Diplopia* develops at times from the restricted movements caused by the proptosis, with or without a paresis of the muscles.

As a rule, Graves's disease shows very slight changes in the fundus. The arteries are usually larger than normal and about the same size as the veins. At times there is a strong arterial pulsation.

ASTHENOPIA, AMBLYOPIA, AND AMAUROSIS OF NERVOUS ORIGIN.

The sight may become weak, partially and even totally lost, without any organic cause, but merely from functional nervous derangement. *Neurasthenia, hysteria,* and *neuroses following injury* may give rise to such symptoms. The hysteric element which underlies these conditions occasions contradictory and confusing reports, and care must be exercised lest such a patient be considered a malingerer. The sight of an hysteric amblyopic eye may be improved by a plane glass, which by psychic effect would remove the inhibition to sight. The ophthalmoscope may show a perfectly normal eye-ground, and no error of refraction may be present.

Neurasthenia.—By exhaustion of the accommodation and insufficiency of the interni muscles the neurasthenic patient, especially when a school-

child, will complain of blurred vision, lacrimation, and headache. Photophobia, visual hallucinations, and photopsia (bright-colored or glittering spots before the eyes) are sometimes experienced. Depression or excitation of spirits, irritability of temper, insomnia, and vertigo are sometimes accompaniments of this condition. In adults the subjective symptoms of neurasthenia are periorbital, supra-orbital, or vertex pains; shooting and boring pains in the eyeballs; and pain on eye-movements. Sensations of heat or cold and dryness of the lids may be noticed. When glasses are worn, the patient may complain of the reflections of light from the edges of the glass or from the frames. The patient may persistently see the tip of the nose.

In nervous asthenopia and hysteria the very effort to see diminishes the acuity of vision and lessens the extent of the field of vision. This is shown in the so-called *fatigue-field.* If the small five-millimetre-square test-object on the perimeter be repeatedly used in the same meridian, and recorded each time *(Wilbrand's method)* it will be noticed that the field becomes more contracted, perhaps as far as ten or five degrees of the fixation-point. Fleeting, island-like defects in the field of vision are characteristic of nervous amblyopia. The functional derangements of vision may temporarily exaggerate the constant defects of sight and of the field of vision which are due to organic disease.

Hysteria.—In hysteric amblyopia the patient may complain of a complete amaurosis of one or both eyes, but usually of but one. Generally amblyopia only is observed, and rarely does complete blindness occur. It is accompanied by a concentric contraction of the field of vision, and a transposition or overlapping of the color-fields, or in some cases complete color-blindness. There may be spasms of the accommodation, blepharospasm, and a paralysis of the orbital muscles. Orientation may be difficult.

Monocular diplopia and *polyopia* not infrequently complicate hysteric amblyopia. They are, however, rarely complained of by the patient, and are elicited only after a careful test. The phenomena are really due to different images being thrown on the retina when the eye is unable to focus while there is a spasm of the accommodation. Each sector of the lens possesses a focal point of its own capable of throwing a distinct and separate image of the same object on the retina.

In all cases of hysteric amaurosis the pupil reacts normally to light. This serves to differentiate it from other forms of blindness. When monocular amaurosis is a symptom, it is often possible to prove binocular vision by the prism test, two objects being seen when a prism is placed before one eye. A person thus tested is not necessarily a malingerer. A malingerer would not present changes in the form- or color- fields or show the fatigue-field. In hysteria there are no ophthalmoscopic changes in the eyeground. There may be an insufficiency of accommodation and a weakness of convergence, when the eye is emmetropic and the ocular muscles are fully developed. The eye-muscles may be paralyzed, or they may be excessively contracted.

Neurosis following Traumatism.—The most marked symptom is concentric contraction of the field of vision, but it is not always present or pronounced. The color-fields show greater variation than the form-fields, but their relative boundaries are not often changed. Both eyes are usually affected. Hemianesthesia when present is usually on the side of the more contracted field. The fatigue-field may be present.

The pupil-reflex is usually normal, but may be wanting. There may be a difference in the size of the pupils. Insufficiency of the internal recti muscles is not rare. Blepharospasm, photophobia, and sensations of light and color may be present.

TREATMENT.—General and nerve-tonic treatment is indicated for all functional nervous disorders. Eye work should be stopped and tinted glasses prescribed. A change of air and scene, exercise without fatigue, bathing, and proper diet should be advised.

OTHER FORMS OF AMBLYOPIA AND AMAUROSIS.

Besides hysteric amblyopia, which has been considered, there are other forms, viz.:—

1. AMBLYOPIA CAUSED BY A DISTURBANCE OF THE CEREBRAL CIRCULATION.—It may be accompanied by scintillating scotomata and concentric defects in the fields of vision. Homonymous hemianopsia and migraine are characteristic concomitant symptoms.

2. REFLEX AMBLYOPIA.—Irritation of the fifth nerve due to carious teeth has produced defects of vision. Intestinal worms, intestinal diseases, disease of the naso-pharynx, and other organic diseases have caused amblyopia. Reflex amblyopia is supposed to be a vasomotor disorder in which the nutrition of the retina or of the central ganglia is affected.

3. CONGENITAL AMBLYOPIA.—Ocular diseases—such as diseases of the optic nerve, chorioidoretinitis, iritis, and glaucoma—may exist in intra-uterine life and permanently affect the sight. Colobomata and an arrest of development may involve the optic nerve and the other parts of the eye or the entire eye (microphthalmos). Ametropia of high degree may cause one eye to go out of use (exanopsia).

4. UREMIC, GLYCOSURIC, MALARIAL, LEAD, DRUG, AND PTOMAIN AMBLYOPIA, and other forms, non-nervous in origin, will be elsewhere considered.

5. PRETENDED AMBLYOPIA AND AMAUROSIS.—If the patient pretends he is blind in both eyes, the detection may be difficult. Feints at striking the face may cause a lid-reflex or a strong light may cause a pupil-reflex, but the latter reflex may be present in a blind eye if the lesion is behind the reflex efferent pupillary nerve-fibres.

TEST FOR BINOCULAR BLINDNESS.—A lighted candle is placed in front of the patient, and a 6° prism with its base out before one eye. If the eyes see, the one behind the prism will move inward, and outward again when the prism is taken away. (Priestley Smith and E. Jackson.)

TESTS FOR NON-OCULAR AMBLYOPIA.—*1. Diplopia Test.*—Have the patient look at an object with a prism base up or down before the good eye. He is apt to say he sees two objects, which is impossible if one eye is blind. The prism must be fully over the good eye, so that monocular diplopia is not produced.

2. The Crossed Diplopia Test.—If a prism of 10° is held base outward before the pretended blind eye, the eye will rotate inward if it has sight.

3. Color Test.—The patient looks at red and green test-type. A green glass is put before the good eye. If he sees the red letters he does so with the pretended blind eye.

4. Harlan's Test.—Place a + 14 D. lens before the good eye, and a — 0.25 D. lens before the pretended blind eye. The good eye is thereby excluded, and, if the patient can see test-type, he does so with the pretended blind eye.

Erythropsia (Red Vision).—Occasionally patients with aphakia and others have red vision lasting perhaps but a moment or a few minutes, and recurring at irregular intervals. It has been noticed in optic-nerve atrophy, glaucoma, and santonin poisoning. The latter may also cause yellow vision *(xanthopsia)*. The cause of such visual disturbances is not positively known, but it is probably an overexcitation of the visual nervous apparatus.

CHAPTER XXIII.

PREPARATION FOR OPHTHALMIC OPERATIONS.

THE successful practice of the operative surgery of the eye requires a combination of factors, all of which must harmonize if the desired result is to be attained. It is necessary to consider the preparation of the surgeon and of his assistants, the preparation of the patient, the advisability of employing an anesthetic, the judicious selection and preparation of instruments, and, finally, the surroundings of the patient during and after the operation.

Qualifications of the Surgeon.—It is assumed that the ophthalmic surgeon is an educated gentleman. He should not be so young as to destroy confidence nor so old and feeble as to jeopard, by unskillful manipulation, the result of an operation. He should have excellent vision. His hand should be steady. He should be a good judge of human nature and should have the art of inspiring confidence. His habits of life should be regular. He should not be given to excesses of any kind. He should be quiet, thoughtful, studious, and attentive. He should be considerate of the feelings of his patient and of his *confrères*. He should be circumspect in his opinions, respectful to his superiors, just to his colleagues, and honorable in his dealings with them and with the public. He should be not too eager to adopt new methods of doubtful utility or too slow to profit by new ideas and discoveries. He should willingly give his time and talents to the poor. From time to time he should publish the results of his observation and experience, in order that other members of the profession may profit thereby. He should frequently visit the clinics of the great masters of ophthalmology to keep pace with the rapid advances which are being made in the science and art of ophthalmic surgery. His assistants should be selected with care and with a view solely to their qualifications.

Advanced age is not a disqualification for a surgeon, provided his hand be steady and his vision good. Owing to narrowing of the pupil, the aged surgeon will require more light than his younger *confrère*. Presbyopia of high amount will limit the surgeon's usefulness, since he can see clearly at only a fixed distance. The disadvantages of age are often more than balanced by the teachings of experience.

If not ambidextrous, the surgeon should become so by practicing upon the eyes of dead persons or on those of animals. For the purpose of acquiring proficiency in this line the enucleated eyes may be fastened in an operating mask (ophthalmophantome), which permits of an extensive

range of movement. The ambidextrous surgeon possesses a marked advantage over his *confrère* who is not thus equipped, and this advantage is particularly noticeable in such delicate procedures as iridectomy and cataract extraction.

Preparation of the Surgeon and Assistants.—Cleanliness is a necessity in ophthalmic surgery. This means that the operator, his assistants, his instruments, and his patient should be aseptic or in a state as nearly approaching thereto as is possible. The surgeon's clothes, his nose and mouth, and his beard (if one is worn) should be clean. Special attention should be given to the preparation of the hands. The following routine is recommended:—

1. Washing with ordinary soap and hot water.
2. The finger-nails should be cleaned.
3. Weir's method of hand-disinfection should be employed.

After washing as above, a tablespoonful of small crystals of sodium carbonate is placed in the palm of one hand, to which one-third as much chlorid of lime is added. After moistening with water, the mixture is to be rubbed into the skin. This should be followed by a rubbing with a sterile towel and by rinsing in sterile water, in bichlorid solution (1 to 3000), or by washing in a solution of permanganate of potassium followed by an oxalic-acid solution for decoloration. The sodium-carbonate lime-chlorid method releases chlorin-gas and reaches recesses of the skin which by other methods of preparation may not be cleared of pathogenic bacteria.

4. After preparing his hands the surgeon should not touch unsterile substances until after the operation has been finished.

Preliminary Preparation of the Patient.—Operations are divisible into those of election and those of necessity. In the former time is given in which to examine the general state of the patient, and to institute such treatment as is necessary to place him in the most favorable condition for operative measures. In the latter class of cases the surgeon has no time in which to institute general treatment. He must be content with determining the state of the heart and great vessels and of the lungs, if a general anesthetic is required.

Wherever it is possible to do so, eye operations should be placed in the class of operations of election. Nearly all cases of cataract, of strabismus, and of lid diseases will naturally fall under this division, while many cases of injury and of glaucoma will require immediate intervention.

Rheumatism, gout, diabetes, albuminuria, arteriosclerosis, bronchitis, cold in the head, alcoholism, epilepsy, and cardiac and hepatic affections should be treated systemically and systematically for some time before operation. If the patient is of advanced age and is decrepit, the administration of tonics and stimulants will be required.

While age in itself is not a barrier to a successful operation, the prognosis in decrepit individuals is less favorable than in those who are stronger. Old people should not be permitted to lie on their backs for long periods,

since hypostatic pneumonia may develop. Any general or local septic condition which may be present should receive attention. The same is true of skin diseases, particularly those which cause itching.

Search should be made for hemophilia. If the patient is a "bleeder" or comes from a family of bleeders, the fact will not only have a bearing upon the prognosis, but will influence the choice of operative procedures. The hemophile should be given a course of preparatory treatment.

Attention to the condition of the throat and nasal chambers of the patient is of importance. If pharyngitis, tonsillar enlargement, or rhinitis be present, either in acute or in chronic forms, it will be advisable to institute a course of local treatment before proceeding to operate upon the eye or its adnexa. The state of the lacrimal passages is of great importance in any case which requires opening of the eyeball. If dacryocystitis is present it should be thoroughly treated long before the execution of any operation upon the globe.

The evening before the operation the patient should take a bath, and, if a male, should be shaved. The epilation of the cilia a few days before the operation has been advocated by Schioetz and Hjort. This practice has not met with favor. Some surgeons, for the purpose of determining whether the conjunctiva is inflamed, apply a test bandage the evening before operation. This procedure is valueless, unless, as in the method of Schmidt-Rimpler, care is taken to inoculate the cornea of a rabbit with the accumulated secretion.

Time for Operation.—Eye operations are performed successfully in all seasons. It will be best, however, to avoid the excessively hot or extremely cold periods. The spring and fall are the seasons which find favor with the majority of ophthalmic surgeons. Operations can be made at any hour of the day or night, provided skilled assistants and proper illumination can be secured. Many ophthalmologists operate in the morning. Perhaps an equal number will prefer the afternoon. It has long been the author's habit to operate in the latter part of the day, both for personal comfort and for the reason that the patient will then have but a few hours of discomfort, sleep soon occurring. This is particularly advantageous in the case of cataract patients, who, if operated early in the day, are likely to rest badly the first night.

Preparation of the Region of Operation.—Although the sterilization of even limited areas of the conjunctiva is impossible, the necessity for careful preparation of the field of operation is not the less important. The manner of preparing the visual apparatus for an operation will vary according to whether the globe and its adnexa are normal or are in a state of inflammation.

THE GLOBE AND ADNEXA ARE NORMAL.—A few hours before the operation the skin of the forehead, cheeks, and eyelids is to be washed with soap and warm water; then with alcohol or ether, to remove fatty substances; then with water, and finally with bichlorid solution (1 to 2000). The con-

junctiva is to be flushed with sterile water or with a sterile physiologic salt solution. Particular attention is given to the skin of the lid-margin, about the roots of the cilia. Some of the French surgeons are in the habit of applying an oily solution of the biniodid of mercury (1 to 2000) to the ciliary margin. It is advisable to remove the secretions of the sebaceous and Meibomian glands by compressing the margin of the lid between the thumb and fingers.

THE GLOBE AND ITS ADNEXA ARE INFLAMED.—Careful search should be made for inflammation of the lid-margins, of the conjunctiva, of the lacrimal passages, and of the nasal fossæ. If time permits, any disorder of these parts should be treated before proceeding with the operation. A simple epiphora is not a bar to intervention, but it is necessary that a lacrimal probe be passed into the sac and upper part of the duct and that the passages be irrigated with bichlorid solution. Acute diseases of the adnexa are rapidly improved by appropriate measures; others, on the contrary, require a long course of treatment. In case of absolute urgency a dacryocystitis may be treated by the excision of the sac, or the canaliculi may be obliterated by means of the galvanocautery. If the operation admits of postponement, it will be wise to treat the inflamed tissues for several days or weeks.

Assistants.—For most ophthalmic operations only one assistant will be required. His duties will consist chiefly in anesthetizing the eye, in mopping up blood or secretions, and in passing the instruments to the surgeon. In complicated procedures, such as resection of the outer wall of the orbit, the removal of tumors which involve the adjacent parts, excision of the sympathetic nerve, and extensive plastic operations, several assistants will be required. One will be needed to give the general anesthetic, another to handle gauze mops which are used to remove the blood, and a third to pass the instruments to the surgeon. In such procedures as the extraction of cataract or the performance of iridectomy one trained assistant will suffice. The anesthetist should be selected with care, and should attend strictly to the giving of the anesthetic and to nothing else. He should be familiar with the treatment of the accidents which sometimes occur from the administration of chloroform or ether.

Preparation of the Instruments.—All large instruments should be washed with soap and water and brushed before and after using. Fine instruments, such as cataract-knives and needles and keratomes, should be dipped in water and dried with gauze, care being taken not to injure their blades. Scissors, knives, keratomes, cystitomes, and iris-forceps should be sharpened at short intervals. All instruments should be made of metal. They should be purchased of a reliable manufacturer. Cheap instruments should not have a place in the armamentarium of the ophthalmic surgeon. The author prefers the instruments made by Tiemann (New York) or those manufactured by Weiss (London). Cataract-knives and keratomes should be carefully tested upon a trial drum before they are sterilized.

Their points and edges should be perfectly sharp. It will be advisable for the surgeon to keep several extra knives and keratomes on hand. Many an eye has been imperiled because of the condition of the instruments used upon it. As Jackson has said, "Simplicity and good judgment in the planning of an operation and neatness, accuracy, and rapidity in its execution are absolutely essential to ideal asepsis. No routine of chemic antisepsis can compensate for torn, bruised, or otherwise injured tissue in the lips of an operative wound. In cataract extraction it is probable that the highest success is missed more frequently through working with a knife of unsuitable shape, or through applying force to it in the wrong direction, than through having it imperfectly 'sterilized.' It needs to be more generally taught and remembered that no routine can make bungling surgery aseptic."

STERILIZATION BY DRY HEAT.—This method, which is not applicable to delicate instruments, is admissible for the grosser ones. They are placed in an autoclave and are subjected to a temperature of 150° C. If the atmosphere is humid, the box containing the instruments should be heated before it is placed in the sterilizer. If this is not done, the steam will condense on the instruments and will rust them.

STERILIZATION BY BOILING WATER.—This is a simple, but an efficient method. The instruments are placed in water which is gradually brought to the boiling-point, and are kept there for five or ten minutes thereafter. Carbonate of soda or borax (2 to 100) should be added to the water to prevent rusting. After removal the instruments should be wiped carefully with dry sterile gauze. The sterilization of cataract-knives and keratomes is best done immediately before beginning the operation. They should be held in boiling water for one minute. Cystitomes should be boiled for two minutes. If these instruments are boiled for long periods their cutting edges will be dulled.

The author intrusts the boiling of large instruments, together with the gauze to be used during the operation, to an assistant. The more delicate instruments, and particularly those which are to be introduced within the eyeball, are held in boiling water furnished by a portable sterilizer, which is placed near to the operating table.

STERILIZATION BY ANTISEPTICS.—This method is inefficient, and is to be rejected. Chloroform, formol, carbolic acid, and strong solutions of bichlorid of mercury are among the agents which have been employed. They all produce irritation of the conjunctiva. If grease or albuminous substances remain adherent to the instruments the liquid cannot come equally in contact with the metallic surfaces and the sterilization is inefficient.

Preparation of Mops and Dressings.—GAUZE.—In the place of sponges, which were formerly in vogue, ophthalmic surgeons now use small pieces of sterile gauze to mop up blood or secretions. To make certain that the gauze is sterile it should be boiled in filtered water before each operation.

Dressings to be applied to operated eyes should be light and should be

sterile. After any operation which involves opening the eyeball a few layers of moist sterile gauze are to be placed over the closed lids of both eyes and a light bandage should be applied. The use of rubber protective, with or without pads of absorbent cotton, is to be eschewed, since such applications cause too much heat. The author always has the commercial sterile gauze boiled in filtered water before it is used as a dressing. What is known as plain gauze—*i.e.*, not medicated—is sufficient for the purposes of the ophthalmic surgeon. Gauze which is impregnated with iodoform or bichlorid of mercury possesses no advantages over the plain, sterile dressing. In a case requiring skin-grafting a suitable dressing will be gauze which has been wet with a sterile physiologic salt solution.

While a simple dressing composed of gauze and a bandage will suffice for most ophthalmic cases, patients who are restless, disobedient, or irresponsible should have a protective mask placed over the dressing. Ring's mask is a good one. It is made of *papier-maché*. Andrews has designated a shield made of aluminum, and McCoy has invented one which consists of two circular frames made of wire. These are curved so as to present a concave surface to the eye.

BANDAGES should be made of soft material,—*e.g.*, of flannel or gauze,—which should be light and porous. They should be sterilized by dry heat. The roller bandage for an adult should be two inches wide and five or six yards long. A bandage for a child should be of a less width. The bandage may be applied to one eye, the ordinary crossed bandage *(monoculus)*, or to both *(binoculus)*. Care should be taken that it is not applied too tightly and that the ear is not distorted. In place of the roller bandage the surgeon may use the bandage of Liebreich or that of Stephenson. The former consists of a knitted cotton or linen band, two and one-quarter inches wide and ten inches long, supplied with tapes. Stephenson's bandage is of dumb-bell shape, and also is provided with tapes.

Anesthetics are divisible into general and local.

GENERAL ANESTHETICS are necessary in major procedures, such as enucleation of the eye, exenteration of the orbit, and extensive plastic operations. They are also required in iridectomy for glaucoma. In children and in nervous adults they may be needed for minor as well as for major operations. Nitrous-oxid gas, bromid of ethyl, ether, and chloroform are the agents used as general anesthetics. Nitrous-oxid gas may be used for operations which can be rapidly performed, such as the dilation of a lacrimal stricture, the opening of an abscess of the lid, or the incision of a panophthalmitic eyeball. Bromid of ethyl—which finds favor with some European surgeons, but has not come into extensive use in this country—possesses no advantages over ether or chloroform. Ether is to be preferred to chloroform, provided the patient's lungs and kidneys are normal. Chloroform is extensively used in operations upon children.

LOCAL ANESTHETICS.—Cocain, eucain, holocain, and tropacocain are the agents used. They are employed in three ways: (1) by instillation,

(2) by hypodermic injection, and (3) by intracutaneous injection (filtration method of Schleich).

Cocain, the oldest and best known of the group, is employed in solutions which vary from 1 to 10 per cent. in strength. The solution should be made sterile by boiling, and a fresh solution should be prepared for each operation. The glass of the eye-dropper also should be boiled before it is used.

The hypodermic injection of cocain is made by means of the ordinary hypodermic syringe. This method is of especial value in the removal of chalazia and small palpebral growths. It is also employed in trichiasis and entropion operations. Some surgeons extirpate the lacrimal sac and remove superficial orbital tumors under local anesthesia. In all such cases cocain is instilled into the conjunctival *cul-de-sac* as well as injected beneath the skin.

The intracutaneous method of anesthesia is little used by ophthalmic surgeons. A favorite solution for this purpose is: cocain, gr. ss; sodium chlorid, gr. ss; water, ℥ss.

Local Hemostasis. — For the purpose of producing temporary ischemia of the conjunctiva and of the tissues of the anterior ocular segment, various preparations of the *suprarenal capsule* are employed. The dried gland may be used in aqueous solution or a preparation known as *atrabilin* may be employed. Most surgeons prefer a solution of *adrenalin chlorid,* which is used in various strengths (1-10,000 to 1-2000). Soon after instilling such a solution the conjunctiva will appear bloodless. The method is of value in numerous operative procedures, such as tenotomy, advancement, pterygium operations, etc. Some surgeons extend its application to cases requiring iridectomy.

The Operation Room should be clean, of suitable size, and properly furnished. It should contain an adjustable table on which the patient is to lie during the operation. Smaller tables on which to place sterile instruments and dressings should be provided. There should also be hot and cold water. The room should be illuminated by one large window, which preferably should face the north. In order to secure suitable illumination on dark days, gas or electric lights should be provided.

CHAPTER XXIV.

THE HYGIENE OF THE EYES.

By HAROLD G. GOLDBERG, M.D., of Philadelphia,

Pathologist to and Curator at the Wills Eye Hospital; Pathologist to the Ophthalmologic Department of the Philadelphia Polyclinic and College for Graduates in Medicine.

IN the field of preventive medicine—a field that has been accorded so prominent a place in the therapeutics of to-day—there is no more important division than that pertaining to hygiene; and in no branch of medicine is the necessity for the observance of a rigid hygiene greater than in that of ophthalmology. When we consider the almost vital part played by the eyes in the conduct of human affairs, the urgent need for a careful preservation of the sight becomes at once apparent. That this last is influenced largely by the maintenance of a proper hygiene there can be but little doubt.

The care of the eyes should begin at birth and should continue uninterruptedly throughout life. There are certain periods, however, when special attention is required. These will be dealt with here in their natural order.

Practically speaking, a child's future—its education, its livelihood, the appreciation and enjoyment of all the beauties of creation—depends upon the preservation of good eyesight. In spite of all the distress and discomforts that attend a faulty vision or a loss of sight, many children are yearly deprived of this faculty simply through neglect to observe the common laws of hygiene. Carelessness in regard to cleanliness at the time of birth, reckless exposure of the infant's eyes to light, pernicious school influences—all these and many other violations of the laws of hygiene in regard to the eyes are largely responsible for the many eye diseases now in our midst. These laws will now be briefly discussed.

THE CARE OF THE EYES AT BIRTH, DURING INFANCY, AND IN EARLY CHILDHOOD.

In caring for a newborn child not only is the observance of the general rules of asepsis from the obstetrician's viewpoint required, but other matters of equal importance are involved. The slightest inflammation of the eyes should be viewed with suspicion,—particularly if the mother gives a history of vaginal discharge,—and should be treated according to indications. No injury can result from the proper application of the ordinary

methods of prevention usually prescribed at this period, whereas the gravest results may follow a neglect to observe every precaution in this regard. The statistics of to-day, compared with those of the past, bear testimony to the wisdom of this procedure.

The infant's eyes should be protected from intense light, and the room should never be brilliantly illuminated, although a judicious amount of sunlight, if properly shaded and not permitted to fall directly into the child's eyes, is wholesome and will result in no injury.

The head of the crib should be turned toward the window or be shaded by a canopy. When the child is taken for an airing, the carriage should be provided with a sunshade lined with a material of a light-absorbing color, such as green, and not, as seems to be the prevailing tendency to-day, shaded by a white parasol, or perhaps not shaded at all.

A child requires change of scene and varied amusement, and for this reason it is most unwise to keep a little one confined to the play-room day after day. It should be remembered that the health of the eyes is directly dependent upon the condition of the general system, and if the aforesaid practice is continued for any length of time, the eyes will suffer.

Children should not be given small toys or picture-books with which to amuse themselves. Often this rule is not easily carried into effect, and if this is the case, their use should be limited to as brief a period of time as possible. In fact, in pleasant weather it is well to insist that children be out of doors as much as possible, for thus they not only receive the benefits of fresh air, but they will also enjoy the constantly changing scenes and pleasures; here, too, close vision is not often required.

Kindergartens, now so popular for the instruction of the very young, are a prolific source of injured eyes. Children who attend these schools are often obliged to amuse themselves with occupations that have a most disastrous effect upon the eyes. These institutions often hold their sessions in buildings never intended for that purpose; frequently they are poorly lighted and too often improperly ventilated. Situated, as they frequently are, in fashionable districts, it is assumed that here the children of wealthy parentage may be instructed in the rules of decorum. Unfortunately, this system is not limited to the favored classes, but has gained in popularity, and has even obtained access into the public schools, where, of course, a larger field for injury presents itself. Myopia and strabismus, in particular, as well as many other morbid conditions, are undoubtedly often the result of the pernicious influences exerted by the kindergarten system.

Light.—Inasmuch as great harm is done the eyes by neglect to observe the utmost caution regarding proper lighting, the subject becomes one of special interest. In spite of the great advances that have been made in the world of science in overcoming what was, not so many years ago, almost a barbarism, and in consideration of the conditions under which our forefathers labored in their efforts for the advancement of learning, passing from the feeble tallow-dip to the dim and uncertain oil-lamp, the existing state

of affairs, when it does not receive the aid and benefit of hygiene, is apparently even worse than was that of former days; then, too, the injurious influences attendant upon these advances have increased proportionately by reason of the greater opportunities afforded. Our exertions should, therefore, be doubled in an effort to correct such evils as may arise.

There are, of course, many conditions which, unfortunately, it is extremely difficult to remedy, as when persons are obliged to pursue their daily occupations in the dim light of stores, workshops, etc., amid poor hygienic surroundings. Nevertheless, even when very delicate work is done continuously at the near point of vision and with defective lighting, these same persons will often, at the close of their working-hours, attempt to read a poorly printed newspaper or magazine in a dimly lighted car, and upon reaching their homes resume their reading by a poor light until their eyes, no longer able to respond to the repeated demands made upon them, close in sleep. It would be useless to detail here all the harmful effects that result from these practices.

Nevertheless, in spite of all this, a great many of the existing evils in the home and in other places where the attention to hygiene is often lax are capable of correction. For example, persons should be cautioned against reading by dim light at any time. It is a very common practice to attempt to read after dusk, by deficient artificial light, and often by moonlight, which is, of course, never sufficiently strong for reading purposes. Bright lights should also be avoided, and, although persons are more apt to err in the other extreme, only direct sunlight need be excluded. Reflected lights, as on the water, or in winter when snow is on the ground, are often very irritating to the retinæ, and under such circumstances dark glasses should be worn.

When we consider that the definition of an object depends upon the amount of light that reaches it, the importance of sufficient illumination, when required, is rendered more evident. The object looked at practically diminishes in size when the light that reaches it is lessened in amount. The inevitable result is that the object is brought closer to the eyes or the eyes to the object, as the case may be. This, of course, requires an undue effort of accommodation and convergence.

Although there is no light that is an actual substitute for direct daylight, the ingenuity of man has devised many useful substitutes. A large number of prismatic devices have been proposed, nearly all having in view the same object, and differing only in construction. Their purpose is so to bend the rays that the light may be distributed equally to all parts of a room instead of being confined to the region of its reception. This is generally effected by the arrangement of a series of prisms that will reflect the light at any desired angle. These contrivances have been made in the form of ornamental windows and shutters that serve not only the aforementioned purpose, but also take the place of the ordinary shutter.

Although this subject is probably deserving of more consideration than has heretofore been accorded it, artificial illumination, by reason of its more elastic practicability and ready adaptation to almost any circumstance, demands our attention. The light that most nearly approaches daylight is the one that serves the best purpose. No attempt will be made here to describe in detail the various merits or faults of the numerous methods of artificial illumination, but mention will be made of a few points common to the most useful of them.

The ideal means of artificial illumination, and the one that is the subject of most of the experiments in lighting to-day, is *fluorescence;* this has the property of giving light with the production of little or no heat. For the present, however, we must be content with the various means of obtaining the highest degree of incandescence from such illuminants as electricity, gas, and oil. This much being granted, it becomes desirable to know which of these is the most suitable for any specific purpose.

ELECTRICITY probably furnishes the purest, whitest light of all the practical light-givers, and is one of the most valuable means of artificial illumination at our command. Among many other advantages that it possesses it may be said to give a maximum amount of light with a minimum amount of heat. It does not vitiate the atmosphere of rooms—a very objectionable feature in other popular methods; it is convenient, comparatively safe, and steady in volume. The naked loop of electrically incandescent fibre possesses properties, however, that, if not removed, may considerably impair its usefulness and give rise to conditions under which prolonged near work cannot be maintained. The loop should, therefore, be shaded by an appropriately tinted translucent bulb, which, while it may decrease the brilliancy of the light, will extend the field of illumination over so great an area that but a slight amount of luminosity will be lost.

The incandescent electric bulb has grown steadily in favor, and most large cities, as well as many suburban towns, are equipped for its service. Its cost grows less each year, and because of its convenience it is deservedly a very popular means of artificial lighting.

The naked *arc,* or free metallic fluid, which is the most powerful artificial illuminant for streets, is generally too powerful for use in buildings, and possesses many peculiarities of quality that are very prone to give rise to symptoms of asthenopia in those who use it for prolonged periods.

GAS.—This, the most common artificial illuminating power in cities, has proved very valuable, and gives evidence of still further usefulness for many years to come. Its disadvantages, however, are that it vitiates the atmosphere, generates excessive heat, and, with old-fashioned burners, gives a yellow light; its volume, too, is affected by the slightest draught. With modern burners the value of gas has been greatly enhanced by the invention of the incandescent mantle; the light thus produced very nearly approaches electricity in quality, giving quite a pure white light.

KEROSENE, the most popular method of artificial illumination in rural districts and among the poorer classes in cities, although less convenient than electricity and gas, may be said to be the least injurious of all the illuminants. Used in the high-grade, modern lamps, it furnishes the most comfortable working light. The lamp should be placed in a well-ventilated room, in a proper position, and be correctly toned and well diffused. Oil has the disadvantages of giving out heat and vitiating the atmosphere. The odor of burning lamps and the trouble incident to keeping them clean are added disadvantages.

Print.—The inferior quality of literature that children are often obliged to read, particularly during school-life and at the age when cheap fiction is a source of amusement, is one of the most prolific sources of eye trouble in later life. Unfortunately, although this period is the most susceptible to improper influences, the trouble does not end with youth, but continues throughout the life of the individual. Newspapers, the most generally read of all literature,—read by all classes and under all circumstances,—are striking examples of poor print.

Taking into consideration the harmful effects of poor printing on the eyes of both old and young, it readily becomes apparent that the proper printing of school-books is a subject of the utmost importance, since at the school-age many maladies may arise from neglect—diseases that could have been prevented by the observance of a few simple rules. School-books should be of a size that can easily be handled, and the type should be large and distinct. It is not easy to fix a definite size for the letters, as so much depends upon their kind and form. If the print is poor and indistinct, the book must be held closer to the eyes in reading, and for this an extra effort of accommodation is demanded; even if the type is large, if it is not printed distinctly the eyes will suffer. Books intended for the use of children should, however, be printed in bold-faced type, with black ink of good quality; a suitable size of type is that known to printers as ten point, or long primer, but if the face of the type is sufficiently heavy, eight point, or brevier, may safely be used. Leading—*i.e.*, the spacing between the lines—and spacing between the words are important aids to the eye; the lines should be at least two millimetres apart. As the eyes move constantly in reading, the external muscles may suffer if the lines are too long; for this reason wide pages should be made up of two or more columns, the lines not exceeding four inches in length, and the columns separated by a space or a rule sufficiently distinct to avoid confusion. The paper should be of good quality, and thick enough to obscure the opposing page. The surface should be dull, and it has been proposed by some that tinted paper be used.

Reading.—The eyes, particularly in youth, are greatly injured by the demands made upon them by an inordinate amount of incessant and laborious work, the most constant of which is reading. The visual organs are no better qualified for constant work than is any other organ of the body, and yet how much oftener are they abused! If an individual is fatigued by

walking, he will, when the opportunity offers, rest; if his arm is wearied, he will pause in his work; but it apparently never occurs to some persons that the eyes are just as susceptible to fatigue as is any other part of the body, and they are thoughtlessly forced into action until, sooner or later, the sufferer is obliged to consult an oculist, who will, of course, attempt to correct the refractive error, if such exists. Very often, however, he will neglect to ascertain the root of the trouble. Frequently, also, thoughtless parents, ambitious to see their children shine in school, in their ardor urge prolonged study upon them. Aside from the injury done to the eye itself, the brain after a time becomes confused by the close application to reading, and although the child may be able to recite his lessons just as he has studied them, he frequently does not comprehend what he has learned to recite. More valuable and humane would it be if children were given more oral instruction, their lessons being expounded to them by means of objects and illustrations placed at their far point.

Later in life the eyes should be spared all the continuous work possible, and frequently should be rested by shifting from one occupation to another, or by closing the eyes at regular intervals for a few minutes during the working-hours, in order to allow the ciliary muscles to relax. Reading matter that possesses the qualities already described as suitable should be preferred; and care should be taken in reading that the book or paper be not held too close to the eyes, for, as has been previously said, this requires an extra effort of accommodation and convergence. Print that cannot be read distinctly at a distance of twenty inches should not be read continuously. Proper illumination is of paramount importance. Light should come preferably from the left side, over the shoulder; otherwise shadows will be formed by the head, shoulders, or hand, according to the faulty relation of the light. Light situated in front and at a considerable distance, unless sufficiently high, should always be avoided, as this mode of receiving light is the most injurious possible. In large office buildings, however, it often becomes necessary to supply each worker—seated at a desk, for example—with an individual source of illumination. When this system prevails and when the light must be in front of and close to the consumer, it should be provided with a shade that will diffuse the light upon the object in front of the worker, at the same time preventing the glare from entering the eyes. If this is impracticable, an eyeshade should be worn, which will usually secure the same result.

The reader should sit upright, his head erect, holding the book almost on a level with the eyes; if, owing to the size or weight of the book, this is not possible, it should be placed on a table or stand, in such a position as to procure the best result. Stooping postures give rise to congestion of the ocular tissues, and the faulty relation of the book to the eyes often causes fatigue of the external muscles of the eye.

Reading while riding in cars is a habit that should be discouraged, for a constant effort of accommodation is required to meet the repeated changes

in focus, and the endeavor of the external muscles of the eyes to keep these organs fixed upon the lines is very fatiguing; eyestrain is often engendered by this all-too-common habit.

A practice equally as injurious to the eyes as the one just mentioned is that of reading while in the recumbent posture. The desire to read during convalescence is sometimes irresistible, but should never be encouraged; at this time the muscles usually are weak, resistance of the tissues is below the normal, and the result of the imprudence may be disastrous. Even in health the practice should not be indulged, for it is almost impossible, without considerable effort, to hold the book in a comfortable position without tiring the external muscles of the eyes; moreover, it must be remembered that the recumbent posture favors congestion—a condition, of course, that were better avoided.

Reading while one is drowsy also has a tendency to promote congestion by forcing the ciliary muscles to action when they would relax, and the external muscles to work when the eyes would diverge.

CARE OF THE EYES DURING SCHOOL-LIFE.

That practically the very existence of a school is dependent upon the hygienic conditions that surround it statistics abundantly show. Of the many evils of a poorly regulated school system, a discussion of which would carry us beyond the limits of this chapter, it may be said that upon no other organ of the body are their influences felt more strongly than upon the eyes. During the period a child attends school the tissues of the eyes may not yet have been fully developed, and it follows that they are less resistant to the morbid influences which they too frequently are called upon to encounter in the school-room. To these influences may be attributed many of the pathologic conditions of the eyes that manifest themselves in later life.

With the increased demands that modern educational methods make upon the youth of our land come greater strain and increased effort, and as a natural result the eyes all too often suffer from the effects of overwork. In the face of all this it must be said that to the present generation is due much credit for improved sanitary conditions and a desire—that in the course of time doubtless will be realized—to bestow upon our growing youth the highest degree of educational perfection with the least expenditure of physical well-being.

In qualifying children for school-life the first question that presents itself for consideration is: At what age are children most fit, mentally and physically, to have thrust upon them the duties of the school-room? Obviously there can be no working rule that can be applied uniformly to every case. As in every other avenue of life in which the regulation of the lives of others is a feature, the individual, and not the system or class, demands our consideration. Into this question should enter the condition of the child physically—in particular, as to the state of the eyes and as to its

mental adaptability. It is no uncommon thing for children utterly unfit—by reason of ill health or faulty vision—for school duties to be permitted to attend school; and these unfortunate little ones are often accused of mental apathy by the careless instructor or thoughtless parent.

As a general rule, children are not fitted to enter school until their eighth or ninth year. Ordinarily at this age the ocular tissues are well formed, and if the child be otherwise physically eligible, he may enter upon a well-regulated school course. With those unfortunates, however, who are afflicted with some high refractive error, corneal opacities, fundus changes, or other infirmity, mental instruction should be deferred and constant use of the eyes eschewed until such time as returning health may direct, and not until every precaution has been taken to correct any refractive error. Increased age brings increased resistance of the ocular tissues, and no harm can follow the delay of a year or two in sending the child to school. In the meantime instruction may be given at home or in special regulated courses at school. Outdoor exercise should be strongly advised.

The importance of detecting a refractive error and making the needed correction cannot be urged too vigorously. The ambitions, the aims, and the very conduct of the sufferer's life are all involved. The development of his character and the molding of his tastes and proclivities, those features which will determine his usefulness in later life, are matters that are implicated.

The problem of determining upon some rational method for qualifying children for school-work now presents itself. It is not necessary for our purpose that a new political office be created, or that a skilled oculist be elected to fill such office. This duty lies chiefly with parents. When a child is deemed ready for school he should be taken to an oculist, who will advise as to his eligibility. Teachers should be instructed in the methods of testing vision, and any child whose visual acuity is found to be below normal should be rejected. Moreover, were teachers obliged to make these tests yearly, it would not rarely be found that those pupils who, at the previous examination, gave slow and hesitating answers, were at this time unfit for the increased and more difficult studies of the succeeding class. These children should receive instruction at home, or their school sessions should be shortened until returning health permits a resumption of the regular class-work. These methods have been adopted with gratifying results in some schools, simple test-cards having been arranged for this purpose.

Children with inflamed eyes or reddened eyelids should not be admitted to school except upon the advice of a physician. The importance of this is made manifest when the danger of infection at toilet to which children in large schools are subjected is considered.

The Construction of School-buildings in Relation to Hygiene.—The construction of school-buildings involves many questions of vital importance. Great care is required in the selection of the location of the building and in securing proper ventilation, drainage facilities, lighting, etc.

LOCATION.—The location of the building should be so chosen as to secure the best possible hygienic surroundings. In cities the building should not be situated on a narrow street, where sufficient light and ventilation are impossible. Investigation has shown that defective eyesight is more prevalent in schools situated on narrow streets and where proper illumination is interfered with by high walls, and it has even been observed that pupils on the lower floors suffer most from defective vision; hence the value of the correct appreciation of this step. The building should be remote from large factories where distracting noises are likely to be heard and where offensive odors and irritating gases are often prevalent. Provision for suitable recreation is highly necessary, and districts where large playgrounds can be provided for out-of-door exercise are to be preferred; when this is impracticable on account of a lack of sufficient space, a large play-room well lighted and properly ventilated should be provided for the amusement of the little ones. This will also serve for use on rainy days.

LIGHT.—The building should be so constructed that light will enter directly, and not be reflected by surrounding walls or windows. All parts of the room should be evenly and diffusely lighted by skylight. It is impossible to err on the side of having too much light, for this can always be controlled. Javal insists that every portion of the room should be so flooded with light that the darkest places will have sufficient illumination on a dark day. A room of oblong shape is usually lighted better than a square room, and should receive its light from windows situated at least four feet from the floor, so that the light will fall above the heads of the pupils. The light should come preferably from the northern side of the room, the northern light being the most constant; when, however, sunlight can reach the room by a different exposure, this advantage must not be disregarded. Intense light may, of course, be controlled by shades or awnings. It is difficult to fix any uniform dimension for the comparative size of windows, as a great deal depends upon the direction from which the light enters and upon the height of surrounding buildings; in any event, the proportion should not be less than one to five.

The walls and woodwork of the school-room should be painted in some light-reflecting color, such as light green, blue, etc. Since the direction of the light is just as important as the amount that is received, care should be taken that the desks be so situated that the light enters from the left and rear; otherwise shadows will be formed by the writer's head, shoulders, or hand; under no circumstances should the light come from the front or fall directly into the face of the pupil.

In working upon an object, it should be remembered that all light that does not come from it can be but a source of annoyance—by irritating the eyes if it is direct, or by confusing the image of the object with the images of other objects if it is reflected. When light comes from the right side as well as from the left, annoying shadows and perverse lights will be formed. At times this direction for receiving light cannot be avoided in

order to allow sufficient lighting and ventilation, and when this is the case, the windows should be placed so high that the light may be diffused, as much as possible, by the ceiling. Even when this has been done, shadows will be formed.

GENERAL PLAN OF THE SCHOOL-ROOM.—The following dimensions, given by Risley, are adapted for the average school-room, and may be used as a general working basis:—

	FEET.
Height of ceiling	15
Length of room	32
Width of room	24
Pier, or blank wall, rear of room	4
Pier, or blank wall, front of room	4
Space allotted to group of windows	24
Windowsill from floor (beveled)	3
Top of window from floor	14

DIMENSIONS OF WINDOW.

Height	11
Breadth	24

Window casing beveled to six inches from ceiling.

FURNITURE.—One of the most important features that affects the comfort and well-being of school-children is the proper construction of the school-furniture.

Desks and Chairs.—The dangers that attend the faulty erection of the school writing-desk cannot be too strongly dwelt upon. It is obvious that to build desks of a uniform height for the use of children of varying sizes must prove harmful. When the seat is too high or the desk too low, the child must stoop while at work, and the upright position cannot be maintained. This is true also if the desk is placed too far in front of the seat, for here again the pupil must stoop over in order to reach the object on which he is at work. On the other hand, if the desk is too high for the seat, the shoulders are forced upward, in which case the arms do not rest upon the desk in the proper position.

Desks should be so built that when the foot rests squarely upon the floor the forearms will rest comfortably upon the top, without forcing the shoulders upward or compelling the child to bend over. To accomplish this the edge of the desk should be on a line with the seat, the back of which should be curved slightly forward; the top of the desk should slope slightly downward—about ten degrees toward the pupil. The seat should be as wide as the thigh is long, measured from the back to the inner bend of the knee. It should be level, slightly grooved to prevent sliding, and low enough to allow the foot to rest squarely upon the floor. A better way would be to build desks so that they may be adjusted mechanically to the requirements of each individual. If desks and chairs are not properly constructed in relation to the size of the individual pupil, spinal deformities are apt to follow. The stooping posture, moreover, favors the gravitation of blood,

and congestion of the ocular tissue and the many evils that attend close vision follow.

In spite of all precautions, many children yet exhibit a marked tendency for near-sight. To overcome this many contrivances have been devised from time to time, most of which consist of a rest so constructed as to hold the head at the proper reading-distance. These contrivances have occasionally proved very useful.

Blackboards, maps, and other objects used for purposes of instruction should be large enough to be seen distinctly from any part of the room, so that pupils will not be obliged to strain their eyes in an attempt to discern the characters. They should be placed at the proper height (about on a level with the eyes), and opposite the source of light, so as not to render the objects obscure by diffusion. Blackboards should be washed with a sponge and water, and not be allowed to become gray under the use of the ordinary eraser.

THE WORK IN THE SCHOOL-ROOM.—As has been stated elsewhere, books should be of a size that can be easily handled, and may be made according to the directions previously given, except that the type may be larger where the occasion offers.

Writing.—This subject has given rise to considerable discussion, but the consensus of opinion seems to be in favor of the use of the vertical script in the school-room, as this is believed to encourage the upright position. The paper should be placed centrally in front of the writer, the ruled lines on the paper being parallel with the plane of the body. The surface of the paper should be dull, or, as has been proposed, tinted, and black ink should be used.

Slates should be entirely discarded, for, besides being uncleanly, the written characters are, for want of contrast, indistinct.

The work in school should be carefully regulated, and frequent intervals of rest allowed; the occupation should be varied from time to time, and it is well occasionally to conduct the instruction by means of conversation, demonstrations, and lectures. Final term examinations and prize competitions should be discouraged; these contests usually occur at the end of the term, when the child is least prepared physically for them. In place of such examinations and competitions the average standing of the pupil in the class and the teacher's report of the student's work during the term, ascertained by unexpected quizzes, should be considered in qualifying the pupil for promotion. Here, again, stress must be laid on the importance of obtaining a plentiful amount of out-of-door exercise for the child; in fact, it is absolutely necessary for the health of the learner that a certain time each day be spent out of doors. Oral instruction is preferable to continuous book-learning, and should be resorted to whenever practicable. Instruction by means of blackboards, maps, and the like is an excellent method of imparting information, for the work is thus accomplished without compelling the pupil to use his near point of vision.

Study at home should be discouraged, for this is often done under the harmful influence of a poor artificial light, with no consideration of the correct relation of light to object, and usually with no attempt to assume a proper posture while at work. Frequently, too, the work is carried on after the child becomes sleepy, an evil that should not be allowed to exist. For similar reasons the reading of cheap literature, in the form of "dime" novels, etc., should be prohibited.

THE PREVALENCE OF MYOPIA IN THE PUBLIC SCHOOLS.

The present system of school-hygiene owes its existence probably to the increase of myopia among the pupils in the public schools. This disease has furnished a basis of construction as to the hygienic requirements of schools. Certainly the disease has been an active incentive to the furtherance of many investigations, for the rapidity with which the malady has increased has been so remarkable and so striking, and the decline after the inauguration of proper school-hygiene so very perceptible, that exhaustive researches have been instigated and many valuable statistics have resulted.

As is well known, the eyes of animals and of uneducated races at birth are hypermetropic, whereas under the same conditions myopia is almost unknown. The statistics of school examinations show that at the beginning of school-life hypermetropic eyes are more numerous than both emmetropic and myopic eyes combined, and with the increase of myopia it has been found that the percentage of hypermetropic eyes decreased proportionately, the intermediate emmetropic eyes remaining at about a standstill. This would seem to show that the change in refraction is a normal evolution; that as the child developed intellectually the eye likewise developed, in order to adapt itself to the increased demands made by the higher education of the individual. To disprove this theory Risley has shown that myopia is not a characteristic of the student any more than of the artisan whose trade requires accurate near vision, provided only that his work was begun early in life. He further contends, in refutation of the theory that the process is physiologic, that in communities where but slight demand is made upon the near vision the necessity for constant care of the eyes is not imperative. He maintains, further, that if children from these communities are sent to school or put at work demanding near vision, the necessity for preventing injury to the eyes is soon made manifest.

As to the pathologic nature of myopia, it is but necessary to review the work of so eminent an authority as Risley. This observer found, in the schools of Philadelphia, that 60 per cent. of eyes with myopic astigmatism presented chorioidal atrophies or inflammations, usually at the temporal margin of the optic nerve; 87 per cent. exhibited varying forms of chorioidal disease; and 70 per cent. were asthenopic.

The pathologic evolution of myopia,—its direct relation to imperfect school-conditions,—although probably well known, may here be briefly

described. Leaving emmetropia out of the discussion, let us assume, for the sake of illustration, a child with hypermetropic astigmatism laboring under the disadvantages of faulty school-influences. In consequence of insufficient illumination, defective print, and the like, the approach of the object to the eyes is made necessary, or, as is more commonly the case in schools, the eyes to the object. This is made easier, at an improperly constructed desk, by stooping. The result is an undue effort of accommodation and convergence, with congestion of the ocular tissues. The globe is pressed upon by the tense external recti muscles, and the tissues become softened by the repeated congestions. Hence elongation of the globe occurs in the direction of least resistance, which is backward. The process is further continued by the resultant myopia, the object being necessarily brought closer for recognition, requiring increased accommodation, convergence, congestion, etc., and so the process continues, elongation and near-sight progressing together. There are probably many more factors in the history of this process, not the least of which is heredity, which is probably responsible for astigmatism; and the various deformities of the skull, which are believed to exert a prominent influence.

THE INFLUENCE OF INJURIOUS HABITS ON THE EYES OF ADULTS.

The influence of pernicious habits upon the general system has its natural effect upon the eyes, which are often not the least to suffer. A discussion of the evils that attend the formation of harmful habits would open up an interminable field of reproach. It is sufficient for our present purpose to mention here the two practices most common to man, and the disastrous effects they exert upon the eyes.

The use of tobacco, notwithstanding all that has been said and written in its condemnation, is, particularly when indulged in moderately, probably not so harmful to the eyes as we are generally led to believe. The smoke is, of course, very irritating to the conjunctivæ, and disease of the optic nerve has been ascribed to the toxic influence. In these cases, however, it will usually be found, upon closer investigation into the history of the case, that there are other causative factors that, if not wholly responsible for the diseased condition, may yet be considered as lending their influence to effect harm. Many persons, moreover, possess a peculiar idiosyncrasy for tobacco.

What has been said of the hurtful influence of tobacco applies in equal degree to the use of alcohol. Even when ingested in small quantities, and likewise when used in the various trades, this idiosyncrasy makes itself manifest.

THE EFFECT OF OCCUPATION UPON THE EYES.

Occupations requiring exposure are not without their ill effect upon the eyes. Among such sufferers may be mentioned cab-drivers, fishermen, etc., who are constantly exposed to violent changes in temperature, to winds,

etc. Chemists, owing to the deleterious effects of the irritating gases sometimes present in chemical laboratories, are frequent sufferers. Machinists are constant victims of flying particles, and every means should certainly be taken to prevent what has long been a prolific source of empty eye-sockets.

While the writer was an interne in the Wills Eye Hospital of Philadelphia more than 2000 cases of foreign bodies in the eye came under his care in one year. Of this number, 90 per cent. were men engaged in occupations in which the eyes were exposed to flying chips of steel, emery dust, fragments of stone, etc. Hundreds of eyes are lost yearly in this way—a fact doubly to be deplored when it is remembered that the wearing of protective glasses would furnish an excellent means of prevention. There are many other occupations through which the eyes suffer, and among such may be mentioned those of dressmakers, watchmakers, engravers, proofreaders, etc.; in fact, as has already been stated, any occupation that requires constant near vision will prove injurious.

Nystagmus may be acquired by the pursuit of certain occupations, and is frequently observed in miners—miners' nystagmus. Nystagmus may also occur in others whose duties require them to keep their eyes fixed in abnormal positions for prolonged periods.

CARE OF THE EYES DURING EPIDEMICS.

There are many infectious diseases of the eyes the spread of which could be prevented were the ordinary rules of cleanliness strictly observed. In the time of an epidemic, or when an infectious disease is known to exist in a household, it becomes the duty of the physician to make known its presence, in order that he may, so far as lies in his power, protect others from infection. When the disease presents itself, the patient should be isolated and provided with toilet articles, bed-linen, etc., for his individual use. If but one eye is infected, the patient should be warned of the danger of autoinfection. All substances used about the affected eye should be destroyed—burned, wherever possible.

THE CARE OF THE EYES IN OLD AGE.

In the declining years of life the tissues of the eye—like those of other organs of the body—become less resistant to disease, and, as a result of undue strain, impaired nutrition, or a faulty hygiene, often become the seat of serious diseases. One of the most common of these is cataract. This affection is rare among those who in youth exercised proper care of the eyes, and who, when the necessity arose, were fitted with suitable lenses. Once the disease has become complete, operative procedure is the only resource. With the observance of a rigid hygiene, and, if deemed advisable, the adjustment of proper correcting lenses, the progress of the disease may in many cases be retarded and amelioration of the symptoms sometimes follows.

As life progresses the power of accommodation gradually diminishes until, when the age of forty or thereabouts is reached, it has almost disappeared. At seventy-five it is usually entirely obliterated. This failure of accommodation, known as presbyopia, is the natural result of oncoming old age. In this condition, the lens, having lost its elasticity, is prevented from assuming that convexity which is necessary for the act of accommodation. This convexity must, therefore, be supplied by suitable glasses.

In advancing old age the greatest care should be exercised that the eyes be not unduly strained by prolonged application to near work. Strong lights should be avoided as much as possible and every effort should be made to shield the visual organs from the effects of irritating vapors and gases. The eyes should frequently be bathed with a solution of boric acid,—10 grains to the ounce of distilled water,—the solution being applied by means of a pipette or eye-dropper. If glasses are worn, these should be kept perfectly clear by repeated washings. It should be borne in mind that glasses require frequent adjusting; particularly is this so when different glasses are used for reading and for distant vision, the frequent interchange rendering this necessary. (See, further, the section on "The Care of Spectacles.")

CLIMATIC CHANGES IN THEIR EFFECTS UPON THE EYES.

In judging of the effects of climate upon the eyes it must be borne in mind that the eye-tissues, as well as the nasal passages, of some individuals are peculiarly susceptible to atmospheric and climatic changes. It seems hardly necessary to state that a person so afflicted should, wherever possible, take up his residence in a district best suited to his condition. At the first premonition of the approach of an attack he should be advised to change his place of residence.

The effects of climatic changes upon the eyes seem to be felt particularly in hospitals, among patients whose eye-tissues are in a low state of vitality, and hence are more susceptible to such influences. During one outbreak of catarrhal conjunctivitis that occurred at Wills Eye Hospital the writer observed 19 cases develop within eight hours. This outbreak occurred simultaneously on both sides of the house, 12 cases appearing among the male patients and 7 among the female, although there was no possible means of direct communication between the two departments. These attacks occur almost invariably at the beginning of the rainy seasons, particularly in the spring and fall.

It has been noted, during the writer's observations in the Wills Eye Hospital, that, when such an outbreak of catarrhal conjunctivitis occurs among the patients, almost every inmate sooner or later succumbs to an attack of the disease, varying in intensity from a slight catarrh to quite violent symptoms. Hence these outbreaks are regarded with a great deal of apprehension, for, aside from the discomforts produced by the disease, the

progress of the healing process is naturally retarded, and as a result of repeated attacks a chronic inflammation ensues.

The disease generally subsides as suddenly as it appeared. The writer has also observed the marked tendency of keratitis, conjunctivitis, etc., to follow upon climatic changes, and he has noted their influence to affect particularly the healing process after corneal sections.

THE CARE OF SPECTACLES.

When the patient has been fitted with glasses, he should be instructed as to the proper method of caring for them, so as to secure the best results. He should be told of the necessity for keeping them adjusted accurately, and the need for cleanliness should also be dwelt upon. It apparently never occurs to some that their glasses require cleaning, and a supposed visual defect will often prove in reality to be but a dimness of vision due to soiled lenses. When such glasses are worn persistently, the straining in an effort to see may give rise to irritation and inflammation of the eyes.

In cleaning spectacles the frame should be grasped firmly close to the hinge, and not at the bridge. The lenses should then be wiped carefully with a clean, unstarched handkerchief, so as to avoid scratching the glasses. At least once a week the glasses should be washed in ammonia-water, except in the case of bifocal lenses, where this operation would be destructive. A fact to be borne in mind is that glasses must be changed at stated intervals; frequently, too, they require changing after prolonged illness or following severe shock.

CHAPTER XXV.

METHODS EMPLOYED IN THE MICROSCOPIC EXAMINATION OF THE EYE.

By W. E. FISCHER, M.D., of St. Louis,

Instructor in Ophthalmology in the St. Louis College of Physicians and Surgeons; Late Voluntary Assistant at the Imperial Royal Eye Clinic of Vienna.

MATERIAL.

To OBTAIN fresh human eyeballs for histologic study is extremely difficult, as comparatively few pathologic conditions require an enucleation of a healthy globe. The chief sources of such eyes are executed criminals, retrobulbar and epibulbar tumors, and resections of the upper jaw. An eye enucleated two to three—at the highest four—hours after death is scarcely to be utilized for careful histologic research.

Pathologic material is more readily procured fresh, as the treatment often necessitates the removal of the globe or the diseased tissue. Some morbid conditions still remain which do not require an enucleation or excision of the diseased tissue and can consequently be obtained only after death.

Enucleation.—For the technique see page 607. The assistant should exercise great caution not to allow the corneal epithelium to dry, nor to brush it off by reckless sponging with dry gauze.

Orientation.—The vessel into which an enucleated bulbus is placed should be labeled immediately after enucleation. Threads may be passed through the tendinous stumps of the internal and external recti muscles. But the form and asymmetry of the bulbus, the shape of the cornea, the stumps of the tendinous insertions of the ocular muscles, the entrance of the two long posterior ciliary vessels, which perforate the sclera on each side of the optic nerve in the horizontal meridian, and the eccentric entrance of the optic nerve will prove of value in orientation.

PRESERVATION OF EYEBALLS FOR DEMONSTRATION.

Dry Method.—Fix the bulbus in formol; then harden and dehydrate thoroughly in absolute alcohol, which should be changed a number of times. Finally the eye is left in pure turpentine for a week, is then taken out, and the superfluous turpentine is allowed to evaporate slowly. Such bulbi will keep indefinitely.

A very good medium is a 4-per-cent. strength formol solution (4 parts of formol to 96 parts of water). The cut surface of the eyeball is laid

on the bottom of a glass container (made by Wall & Ochs). The glass rod in the rubber stopper is pressed down on the spheric side of the eye to hold it in place.

Kaiserling's Method is admirably adapted to preserve the colors and transparency of the ocular structures; it is as follows:—

Formol	750.0
Distilled water	1000.0
Potassium nitrate	10.0
Potassium acetate	30.0

The specimens are allowed to remain in this solution for twenty-four hours. Transfer to 80-per-cent. strength alcohol for twelve hours. They are then placed in 95-per-cent. strength alcohol for two hours. The preparations are preserved in a mixture of water and glycerin, equal parts, with the addition of 30 parts of potassium acetate. Very delicate objects are to remain in this mixture only for from one to two days, and are finally preserved in a mixture of equal parts of water and glycerin with the addition of a little absolute alcohol (1 to 10).

Method of Priestley Smith.—The eye is bisected, preferably after it has been frozen. The yellow stain imparted to a specimen fixed in Müller's fluid may be removed by placing it in a 5-per-cent. strength solution of chloral hydrate, which is renewed every two or three days until the solution no longer becomes tinged.

Now place the preparation successively in 10-, 25-, and 50-per-cent. strength glycerin solutions, allowing it to remain for twenty-four hours in each. Preserve the specimen in a jelly consisting of:—

Best French gelatin (Coignet & Co., Paris)	1.0
Water	6.0
Glycerin	6.0

The gelatin remains in the water until it swells, and is then melted by gently warming; now add the glycerin and a few drops of carbolic acid or thymol. Filter through flannel while warm.

Method of Nettleship.—

Glycerin	250.0
Gelatin	250.0
Creosote	1.0

All the interstices of the preparation are filled with the mixture, and then the air-tight jar is filled with the jelly and sealed.

FIXING.

The object of fixation is to retain the tissue-elements in the condition which the tissue presented in life or at the moment of death, and to prevent further *postmortem* changes. Most of the fixing fluids coagulate the albumin, while others dehydrate.

Müller's Fluid (Introduced by H. Müller).—

Potassium bichromate	2.5
Sodium sulphate	1.0
Distilled water	100.0

The fluid must be used in considerable quantities and renewed daily until it no longer becomes cloudy. For thorough fixation a period of six weeks at the temperature of the room (70° F.) will be required. By placing the specimen in an incubator at a temperature of 36° to 40° C. and changing the fluid daily fourteen days will suffice. To prevent the development of fungi a little camphor or carbolic acid may be added. Virchow recommends keeping the specimen in the dark to prevent the formation of precipitates in the tissues.

When fixation is complete the specimen is to be washed in flowing water for twenty-four hours and then is hardened in alcohol of increasing concentration (see page 777). Müller's fluid enjoyed great popularity among ophthalmologists, but it has been partly superseded by formol, sublimate, and picric acid-sublimate, which are sometimes preferred. A specimen should be fixed, hardened, and stained in accordance with the peculiarities sought for.

Formol.—Formol, or formalin, is an aqueous solution containing 40 per cent. of formaldehyd-gas. It is used in strengths of 4 to 10 per cent. An ordinary working solution is:—

Formol	10.0
Distilled water	100.0

The above solution fixes in from twelve to twenty-four hours.

If the eyeball be left in formol too long the lens and sclera get very hard. A 4-per-cent. strength formol solution is a suitable agent to preserve eyes which are intended for museum purposes.

Formol-Müller Fluid.—

Potassium bichromate	2.5
Sodium sulphate	1.0
Formol	10.0
Distilled water	100.0

Or to 100 cubic centimetres of Müller's fluid are added 10 cubic centimetres of concentrated formol solution. The specimen is fixed in from six to fifteen hours. Wash in flowing water for twenty-four hours. The formol is preferably added immediately before the solution is used, since the mixture loses its efficacy in a week.

Absolute Alcohol is not a good fixing agent for the eyeball, which it shrinks too much. The finer structures are not well preserved. It answers very well where we wish to examine for tubercle bacilli or leprosy bacilli.

Erlitzki's Fluid.—

Potassium bichromate	50.0
Copper sulphate	10.0
Distilled water	1000.0

This fluid fixes more rapidly than Müller's fluid. Ten days at the temperature of the room or four days in an incubator are sufficient. The eyeball is then to be thoroughly washed in water for twenty-four hours. Erlitzki's fluid is similar to Müller's fluid, but it produces more shrinking and more precipitates.

Sublimate.—

Corrosive sublimate, saturated solution,
Alcohol, 95-per-cent. strength.....of each 100.0

The eyeball remains in the solution for twenty-four hours. It is then washed thoroughly in flowing water and finally is transferred to a brown-red solution of iodin in 70-per-cent. strength alcohol, which must be changed until it retains its brown-red tint. The iodin removes the sublimate crystals which were precipitated in the tissues. Sublimate is an admirable fixing agent for the preservation of karyomitotic figures.

Flemming's Solution.—

One-per-cent. strength chromic acid	15.0
Two-per-cent. strength osmic acid	4.0
Glacial acetic acid	1.0

Allow the preparation to remain in this solution for at least twenty-four hours. This is a splendid medium for fixing the retina, the vitreous body, karyomitotic figures, and fat.

Other fixing agents are: picric acid, sublimate-picric acid, 3-per-cent. strength nitric acid, osmic acid, etc.

WASHING.

Specimens that have been fixed in Müller's fluid, in picric acid, or in Erlitzki's fluid should be washed in flowing water for twenty-four hours. The specimen may be placed in a jar into which a small rubber tube hangs from the faucet. If the specimen is small, a piece of gauze may be tied over the top of the jar to prevent it from getting lost. If a hydrant is not convenient, a large jar in which the water is frequently renewed will answer as well.

DECALCIFICATION.

In certain instances calcification or ossification renders the sectioning with the microtome difficult or impossible. In order that sectioning may be done the specimen must be decalcified. Lime deposits may occur in all portions of the eye. Perichorioidal ossifications are very common after inflammations. Calcareous deposits are frequent in gliomata of the retina, and ossifications in sarcomata of the chorioid have been occasionally observed.

The best fluids for decalcification are:—

1. Müller's Fluid.—Müller's fluid will decalcify, provided the specimens be left in the solution long enough. It decalcifies very slowly, and it will be necessary to permit the specimens to remain in it for months.

2. Picric Acid.—Picric acid in a concentrated aqueous solution decalcifies slowly because it does not penetrate into the tissues deeply. The same is true of picro-nitric acid.

3. Method of Haug.—This method is well adapted to decalcify the tissues of the eye, especially if the specimens have been fixed in formol or sublimate.

Nitric acid	3.0 to 9.0	c. cms.
Absolute alcohol	70.0	c. cms.
Distilled water	30.0	c. cms.
Sodium chlorid	0.25	c. cm.

4. Phloroglucin Method.—

Phloroglucin	1.0
Nitric acid	5.0
Alcohol	70.0
Distilled water	30.0

The above solution decalcifies rapidly. The time required for complete decalcification varies with the amount of calcareous material to be removed and the thickness of the specimen. To ascertain if the decalcification is complete, we prick the area of the deposit with a teasing needle. After the calcareous deposit has been removed, the specimen should be washed in water for several days.

If the condition remained unnoticed until the specimen was mounted for sectioning, it should be placed in equal parts of absolute alcohol and ether to dissolve the celloidin and then should be decalcified.

BLEACHING.

In order to render the examination of the minute details of some normal and pathologic ocular tissues possible, the pigment which is naturally present must be removed. Among the bleaching methods are the following:—

Hydrogen Peroxid.—The celloidin sections are washed in water and are then placed in peroxid of hydrogen and are exposed to the sunlight for two or three days until they are sufficiently bleached. The sections become brittle by this process, but they will stain well.

Chlorin.—The chlorid of lime or chlorin-water may be used, but they also make the sections fragile.

Griffith's Method.—

Potassium chlorate	1.0
Concentrated hydrochloric acid	2.0
Distilled water	300.0

The mixture must be shaken from time to time and should remain in the dark.

Alfieri's Method.—1. Place the sections in a potassium-permanganate solution (1 to 2000) for twenty-four hours. When exposed to sunlight, the sections assume the brown color in less time.

2. Transfer the brown sections to a solution of oxalic acid (1 to 300), which fully bleaches them in a few seconds.

The sections will be fragile. After all bleaching processes the specimens do not stain as well as unbleached sections; hence they must be left in the stains longer.

HARDENING.

After the eyeball is fixed and washed and eventually decalcified, it is ready to be hardened. We usually employ alcohol in increasing concentrations for this purpose as follows:—

The eye is placed in alcohol: 70-per-cent. strength for twenty-four hours, 80-per-cent. strength for twenty-four hours, 90-per-cent. strength for twenty-four hours, 95-per-cent. strength for twenty-four hours, and in absolute alcohol for forty-eight hours.

For practical purposes it will suffice if the eye be placed in 70-per-cent. strength, in 90-per-cent. strength, and in absolute alcohol. The percentage of dilute alcohol need not be absolutely accurate. It may be roughly made as follows:—

70-per-cent. strength alcohol = 74 c. cms. of 95-per-cent. strength alcohol + 26 c. cms. of distilled water.
80-per-cent. strength alcohol = 84 c. cms. of 95-per-cent. strength alcohol + 16 c. cms. of distilled water.
90-per-cent. strength alcohol = 95 c. cms. of 95-per-cent. strength alcohol + 5 c. cms. of distilled water.

The absolute alcohol should be changed once after twenty-four hours.

CUTTING THE EYEBALL PREPARATORY TO MOUNTING.

The eye is ready to be cut for mounting when it has been sufficiently hardened. The sections are cut according to the condition for which we desire to examine. The globe may be cut in quadrants (a very convenient form), one section being made in the vertical meridian and the other in the equator. If the lens is to be kept in place, the eye may be capped in a horizontal direction above and below the lens. In cases in which the displacement of the lens is a matter of indifference the bulbus may be divided in the horizontal meridian into an upper and lower half. If a tumor is present, we may divide the globe in the meridian in which the growth is situated.

IMBEDDING.

Celloidin Imbedding.—Specimens that have been hardened and cut must be imbedded in some solid material in order to render sectioning with the microtome possible. Celloidin imbedding, proposed by Schieferdecker, was first applied for imbedding eyes by Becker, and is practically the best method. Celloidin (Schering) is obtained in sealed bottles. Thick celloidin is made by allowing a sufficient amount of celloidin to dissolve in equal parts of absolute alcohol and ether to make a fluid the consistency of thick syrup. Thin celloidin is obtained by further diluting thick celloidin with equal parts of absolute alcohol and ether until it is a thinner, less viscid fluid resembling collodion.

After the globe has been hardened and dehydrated in absolute alcohol it is placed in an absolute alcohol and ether mixture, equal parts, for twenty-four hours. It is then put in thin celloidin for from four days to two weeks. The longer the specimen remains in the thin celloidin, the more thoroughly its tissues will be permeated by it. It is finally transferred to the thick celloidin and left in it for the same length of time, when all is ready for mounting.

For the purpose of mounting, the specimen is placed in a glass vessel deep and wide enough to allow at least one-fourth inch space on all sides. The specimen is put in the centre and the thick colloidin is poured on until it rises one-fourth inch or more above the specimen. The vessel is then covered with a small bell-jar, and the alcohol and ether are allowed to evaporate very slowly in a cool place. The celloidin is permitted to harden until it has the consistency of wax: *i.e.*, to a degree that an impression with the finger-tip (not the nail) can scarcely be made. This usually requires from two to four days, depending on the rapidity of evaporation, etc.

The firm celloidin is loosened by passing a knife along the walls of the vessel, and its contents are expelled by gently tapping on the bottom. The superfluous celloidin is trimmed off, and the specimen (inclosed in celloidin) is then placed in 70-per-cent. strength alcohol for twenty-four hours, when it will be sufficiently hardened to be mounted on a block of wood, cork, iron, or stabilit (insulating fibre) which fits the microtome-clamp. Thick celloidin is spread on one surface of the block of wood or cork and the celloidin block, with its inclosed specimen, is gently pressed on it. After ten minutes the wood and the celloidin block are immersed in 70-per-cent. strength alcohol and left in it for a number of hours, when all is ready for sectioning with the microtome.

Paraffin Imbedding briefly consists of the following steps:—

1. Thorough dehydration of the specimen in absolute alcohol for from five to twenty-four hours.

2. Place in xylol for from one-half to four hours.

3. Place in xylol-paraffin (concentrated solution of paraffin in xylol) for from one to six hours.

4. Melted soft paraffin (42 degrees) for from one-half to two and one-half hours.

5. Melted hard paraffin (58 degrees) for from one-half to two and one-half hours.

6. Place the specimen in a mold and pour on 58-degree paraffin and allow to cool.

7. Mount on a block which fits the microtome-clamp.

The above method is admirable for the retina, chorioid, etc., but if it be used for a globe the lens must be removed from the eye.

Paraffin sections are mounted by spreading a drop of water containing a trace of alcohol on a slide, on which the section is now laid. The slide may be warmed until the water has evaporated; or the slides may be

placed in an incubator and kept at a temperature of 30° to 35° C. for from twelve to twenty-four hours. If the specimen has been fixed in Müller's fluid, chromic acid, or osmic acid, the sections are preferably fixed on the slide with a mixture of the filtered white of an egg and an equal quantity of glycerin to which a few grains of sodium salicylate or a trace of thymol have been added to prevent the growth of fungi.

A drop of this mixture is put on the slide and the technique for mounting the section on the slide is the same as for the water with a trace of alcohol.

The paraffin must be removed from the mounted paraffin sections before they will accept any stain. This is accomplished by placing the slide on which the section has been fastened in a vessel containing pure xylol. Then the xylol should be removed with absolute alcohol; if aqueous solutions of the stains are to be used, the section is next transferred to 90-per-cent. strength alcohol, then to 60-per-cent. strength alcohol, and finally to water, when it is ready to be stained.

CUTTING THE SECTIONS.

The specimen-block should be clamped firmly in the microtome. In cutting specimens that have been mounted in celloidin the microtome-knife should be set at as acute an angle as possible, in order to utilize as much of the cutting edge of the blade as we can. The knife should be drawn slowly and evenly to obtain sections free from tears and of a uniform thickness. The blade and the specimen should be kept well moistened with 80-per-cent. strength alcohol to keep the specimen from drying, and to permit the sections to float on the knife. The specimens may be washed in water and then stained, or they may be preserved in 80-per-cent. strength alcohol.

In cutting paraffin sections the knife is not placed at so sharp an angle and the sections are cut dry. If ribbon sections are to be cut, the knife is set at right angles to the direction in which it moves, and the paraffin should be quadrilateral. The sections are prevented from rolling up and are removed from the knife with a fine camel-hair brush or a delicate bristle. If the sections should be wrinkled, they may be placed in tepid water, when they will flatten out.

STAINING.

NUCLEAR STAINS.

1. Hemalaun of P. Meyer.—One gram of hematein is dissolved in 50 cubic centimetres of 90-per-cent. strength alcohol by warming. This is added to a solution of 50 grams of alum in 1000 cubic centimetres of distilled water. A crystal of thymol may be added to prevent the growth of fungi.

STAINING.—1. Wash the sections in water.

2. Stain for from five to fifteen minutes.

3. Wash in water for from ten to twenty minutes.

4. Dehydrate in 95-per-cent. strength alcohol.

5. Clear in carbol-xylol. Balsam.

2. Delafield's Hematoxylin.—Four hundred cubic centimetres of a concentrated aqueous ammonic-alum solution is mixed with a solution of 4 grams of hematoxylin in 25 cubic centimetres of absolute alcohol. The mixture is exposed to the light in an open vessel for three or four days; 100 cubic centimetres of methylic alcohol and 100 cubic centimetres of glycerin are then added, and the stain is allowed to mature. In a few days it is filtered and is ready for use.

STAINING.—1. Sections are washed in water (or 1-per-cent. strength alum solution).

2. Sections are stained for from two to three minutes; if the stain has been diluted with water, ten to fifteen minutes. The section should have a light-blue tint, not an intensely dark one.

3. Wash in water until no more blue color is given off (for from ten to fifteen minutes to several hours).

4. Dehydrate in 95-per-cent. strength alcohol.

5. Clear in carbol-xylol. Balsam.

If a section has been overstained in hemalaun or hematoxylin, the desired tint may be obtained by the following procedure: Place the overstained section in $^1/_{10}$-per-cent. strength muriatic acid until it turns red, then wash the section thoroughly in water for several hours. Should the section be still overstained, the procedure can be repeated until the proper tint is obtained.

3. Alum-carmin (Grenacher).—Boil 1 gram of carmin in 100 cubic centimetres of a 5-per-cent. strength alum solution for twenty minutes; when the solution is cool, filter it.

STAINING.—1. Wash in water.

2. Stain in carmin for from ten minutes to several hours (this carmin does not overstain).

3. Wash in water.

4. Dehydrate in 95-per-cent. strength alcohol.

5. Carbol-xylol. Balsam.

4. Borax-carmin (Grenacher), Aqueous Solution.—

Carmin	0.5
Borax	2.0
Distilled water	100.0

The above ingredients are mixed and boiled, and 5 cubic centimetres of a $^5/_{10}$-per-cent. strength solution of acetic acid are added, drop by drop, while the mixture is being constantly stirred until it turns to a deep-red color. Filter the solution after twenty-four hours.

STAINING.—1. Wash in water.

2. Stain in carmin for from five to twenty minutes.

3. Differentiate in hydrochloric acid-alcohol (concentrated hydrochloric acid, 1.0; alcohol, 70-per-cent. strength, 100.0).

4. Wash thoroughly in water.

5. Alcohol, carbol-xylol. Balsam.

5. Borax-carmin (Grenacher), Alcoholic Solution.—The alcoholic solution is used if the sections are not to be brought in contact with water. It is prepared as follows:—

Carmin	2 to 3
Borax	4
Distilled water	93

After forty-eight hours the above solution is mixed with 100 cubic centimetres of 70-per-cent. strength alcohol. Allow the mixture to stand for thirty-six hours' time and then filter it. Nuclei stain red with this solution.

STAINING.—1. Transfer the section directly from 70-per-cent. strength alcohol into the alcohol-carmin and stain for from five to twenty minutes.

2. Differentiate in hydrochloric acid, 1.0; alcohol, 70-per-cent. strength, 100.0.

3. Wash in 70-per-cent. strength alcohol for several hours.

4. Dehydrate in 95-per-cent. strength alcohol.

5. Carbol-xylol. Balsam.

6. Lithia-carmin (Orth).—Dissolve 2.5 grams of carmin in 100 cubic centimetres of a cold, saturated solution of lithia carbonate.

STAINING.—1. Wash the sections in water.

2. Stain in the solution for from five to ten minutes. (By warming gently over the vapor of a water-bath two to five minutes will suffice.—Obersteiner.)

3. Differentiate in hydrochloric-acid alcohol as before.

7. Safranin (Pfitzner).—This is an admirable stain for specimens that have been treated with Flemming's solution.

Safranin	1.0
Absolute alcohol	100.0
Distilled water	200.0

Dissolve the safranin in alcohol and then add the water. Stain the sections in this stain from two to twenty-four hours. If the specimens have been fixed in Flemming's solution and the sections stained with safranin and treated with acid-alcohol (8 drops of hydrochloric acid or 10 drops of a concentrated solution of picric acid to 100 cubic centimetres of alcohol), until the celloidin is colorless, the chromatin of the nuclei only will be stained. Absolute alcohol. Xylol. Canada balsam.

Karyomitotic figures are stained intensely red; the nuclei at rest are pale pink. Mucin appears yellowish red, while fibrin takes a deep-red tint.

8. Fuchsin (Rubin).—This stain is prepared and used like safranin.

PROTOPLASMIC STAINS.

The basic anilin dyes stain the nuclei, while, on the other hand, the acid anilin dyes stain the protoplasm diffusely. The latter are seldom employed alone, but are nearly always combined with a nuclear stain.

1. Eosin (Fischer).—

Eosin	1.0
Water	100.0

STAINING.—This solution may be diluted from three to five times with water.

1. Place the section in the diluted stain for from three to five minutes; in some instances one minute will suffice.

2. Wash in water for a few minutes.

3. Dehydrate the sections in 95-per-cent. strength alcohol.

If the section should have been overstained it may be left in 70-per-cent. strength alcohol until the excess of the eosin has been extracted.

One-per-cent. strength eosin in 90-per-cent. strength alcohol may also be employed. Stain the same as above with the exception that the specimen is transferred from the stain directly to alcohol.

2. Picric Acid may be used as a counterstain in a concentrated aqueous or alcoholic solution. In certain instances it may be still further diluted before using. Should the tissue be overstained, the excess can be removed by washing in water or alcohol. If picric acid is used as a counterstain with hematoxylin or with hemalaun, the tissue must be slightly overstained, as picric acid will extract them.

3. Orange G, in a dilute aqueous solution (1-per-cent.), stains the protoplasm light orange.

4. Acid Fuchsin (Fuchsin S., Rubin S.) is used as a concentrated aqueous solution, or it is dissolved in anilin-water. It readily overstains, and this fault cannot be remedied.

DOUBLE STAINS.

The following combinations of nuclear and protoplasmic stains produce good results:—

Hemalaun or hematoxylin and eosin.	Carmin and orange G.
Hemalaun or hematoxylin and orange G.	Carmin and picric acid.
Hemalaun or hematoxylin and picric acid.	Safranin and picric acid.

Hemalaun-eosin is a durable stain and the best for all practical purposes.

STAINING.—1. Stain in hemalaun for from five to ten minutes (see hemalaun).

2. Wash in water for from ten to thirty minutes.

3. Stain in a diluted 1-per-cent. strength aqueous solution of eosin or in an alcoholic solution for from one to five minutes. (The alcoholic solution is composed of: eosin, 0.1; alcohol, 90-per-cent. strength, 100.0.)

4. Wash in water for a few minutes.

5. Alcohol, 95 per cent. Should the specimen be overstained in eosin, it may be placed in 70- to 80-per-cent. strength alcohol until sufficient eosin has been extracted to give the section a rose-red tint.

6. Carbol-xylol.

7. Balsam and cover-glass.

Van Gieson's Stain is a beautiful tricolor stain, but unfortunately it is not always permanent.

1. Overstain section in hematoxylin for from fifteen to thirty minutes.

2. Wash thoroughly in water.

3. Stain for from one to three minutes in a concentrated aqueous solution of picric acid, to which sufficient aqueous concentrated solution of acid fuchsin has been added to give a deep-red color.

4. Wash in water for one-half minute.

5. Alcohol, with a trace of picric acid.

6. Carbol-xylol. Balsam.

The above is an admirable stain for the optic nerve. With this stain colloid bodies assume an orange-red tint, while hyalin bodies are bright red.

Weigert's Stain for Medullary Nerve-sheaths.—Specimens that have been fixed in Müller's fluid, Erlitzki's fluid, or 5-per-cent. strength potassium-bichromate solution are not washed, but are rapidly hardened in alcohol, imbedded in celloidin, and sectioned.

The sections are placed in:—

Saturated solution of neutral copper acetate	50.0
Distilled water	50.0

for from twelve to twenty-four hours.

They are then transferred to a solution composed of:—

(a)	Hematoxylin	1.0
	Absolute alcohol	10.0
(b)	Lithia carbonate	1.0
	Distilled water	100.0

where they remain for from twenty minutes to twenty-four hours. It is preferable to keep the solutions *a* and *b* separate or mix the quantity one wishes to use in the proper proportions immediately before staining.

When the sections are black they are washed and differentiated in:—

Borax	2.0
Ferricyanid of potassium	2.5
Distilled water	100.0

The latter solution may be diluted with water to one-half its strength, so that the process of differentiation may be better controlled. If the staining is successful, the nerve-sheaths will appear dark blue, the degenerated areas, as well as the remaining tissue, light brown. Blood and fibrin often take a dark-blue tint.

Method of Marchi.—1. Fix in Müller's fluid for at least eight days.

2. Transfer to a freshly prepared mixture of equal parts of Müller's fluid and osmic acid for from six to twelve days.

3. Wash in flowing water for twenty-four hours.

4. Harden in alcohol; celloidin. Section with the microtome.

5. Stain the sections in lithia-carmin for contrast.

Degenerated areas appear black, everything else light yellow.

Other methods are those of Paland Kulschitsky. For their execution the reader is referred to the special works of Greeff or Seligmann.

DEHYDRATION OF THE SECTIONS.

Paraffin sections are placed in 60-per-cent. strength alcohol for a few minutes, then in 90-per-cent. strength alcohol, and finally in absolute alcohol.

Celloidin sections are dehydrated in 95-per-cent. strength alcohol, since absolute alcohol dissolves the celloidin. If the celloidin has stained as intensely as the tissue,—for instance, after staining with anilin dyes,—it may occasionally be necessary to remove the celloidin. This is accomplished as follows: The section is treated with absolute alcohol for about five minutes and then is placed in a mixture of absolute alcohol and ether (equal parts) for from ten to fifteen minutes. Oil of cloves will also dissolve the celloidin, but it must be removed again by pure xylol.

CLEARING OF THE SECTIONS.

Paraffin sections that have been thoroughly dehydrated with absolute alcohol are then treated with xylol, in which they will clear in a few minutes.

Celloidin sections that have been dehydrated only in 95-per-cent. strength alcohol are preferably placed in carbol-xylol for clearing. Carbol-xylol is an excellent clearing agent, and is prepared as follows:—

Pure xylol	45.0
Crystallized carbolic acid	15.0

In place of xylol or carbol-xylol, origanum-oil, oil of bergamot, oil of cedar, or oil of lavender may be employed. Oil of cloves is rarely used for the purpose of clearing celloidin sections, as it dissolves the celloidin and extracts most of the anilin dyes.

MOUNTING THE SECTIONS ON SLIDES.

After a celloidin section has been cleared it is spread smoothly on the slide. When all wrinkles have been brushed out with the teasing needle, the clearing medium is blotted off by firmly pressing the section to the glass with several layers of fine filter-paper. Now put 1 drop of Canada balsam on the section and place the cover-glass on it.

For paraffin sections the process is the same, with the exception that the section is fixed to the slide and the process is carried out on the slide.

Sections that are not to be brought in contact with alcohol after they have been stained are mounted permanently in glycerin. The section is placed on the slide, the water is blotted with filter-paper, and then 1 drop of glycerin is put on the section, when the cover-glass is gently placed on it.

In order to keep such a preparation permanently the edges of the cover-glass must be fastened to the slide by rimming the cover-glass with cement. Superfluous glycerin about the edges of the glass must be removed with cotton moistened with alcohol. The cover-glass may be fastened to the slide by painting the edges with melted paraffin over which a coat of asphalt varnish is then applied. Kroenig's cement may be also used.

DEMONSTRATION OF DEFINITE SUBSTANCES AND TISSUE-ELEMENTS.

Nuclear and Protoplasmic Structures.—(A) NUCLEAR STRUCTURES. —*(a)* Flemming's solution. *(b)* Sublimate or formalin fixation should be used. This method is applicable for demonstrating the structure of the nucleus.

Fix in Flemming's solution and imbed in paraffin.

1. Stain in safranin (page 781).

2. Stain in a 2-per-cent. strength gentian-violet solution. Differentiate as with the safranin.

3. *Gram's Method (see page 791).*—Anilin-water and gentian-violet (from three to five minutes). Treat with a solution of iodin, 1.0; potassium iodid, 2.0; water, 300.0, for from one to two minutes.

4. Decolor in absolute alcohol.

5. Stain in carbol-fuchsin:—

Five-per-cent. strength aqueous carbolic-acid solution..	100.0
Fuchsin	1.0
Alcohol	10.0

Stain in this solution for one-half hour, then rinse in 90-per-cent. strength alcohol.

6. Wash in hydrochloric acid-alcohol (see page 781) or 1-per-cent. strength picric-acid solution for from one-half to two hours.

7. Wash in absolute alcohol until only slight stains are imparted to the alcohol.

Clear in xylol. Balsam.

Karyomitotic figures are intensely red; nuclei at rest have a pale-red tint.

(B) PROTOPLASMIC STRUCTURES.—Altmann recommends the following method for the demonstration of the granules in the cell:—

1. Fixing in a solution of:—

Five-per-cent. strength potassium bichromate,
Two-per-cent. strength osmic acid.....of each, equal parts.

2. Wash in water and harden in alcohol of increasing concentration.

3. Imbed in paraffin. Cut sections three to five micromillimetres. Mount and remove paraffin.

4. Stain in a solution of:—

Anilin-water	100.0
Acid fuchsin	20.0

Warm until the solution begins to steam.

5. Allow to cool and remove the surplus of the stain with a mixture of:—

Concentrated alcoholic solution of picric acid	1.0
Water	2.0

Renew the picric-acid mixture and keep the same at a temperature of 42° C. for from thirty to sixty hours.

Wash in absolute alcohol, xylol, and balsam.

The red granules are easily recognized in the pale-yellow cell-body.

Elastic Fibres.—METHOD OF WEIGERT.—

One-per-cent. strength aqueous solution of fuchsin rubin	100.0
Two-per-cent. strength aqueous solution of resorcin	100.0

are mixed in a porcelain dish and brought to the boiling-point. To this solution are added 25 cubic centimetres of liquor sesquichlorati (Ph. G.) and the mixture is boiled for two or three hours. A precipitate is formed. The solution is allowed to cool and is then filtered; the filtrate is rejected. The precipitate on the filter-paper is now boiled in 200 cubic centimetres of 94-per-cent. strength alcohol; the solution is again allowed to cool, and finally is made up with alcohol to 200 cubic centimetres; 4 cubic centimetres of hydrochloric acid are added, whereupon the stain is ready for use. This stain may be purchased of Dr. Grübler, in Leipsic.

Liquor ferri sesquichlorati is made as follows: 1 part of iron is dissolved in 4 parts of hydrochloric acid, and then there are added, for every 100 parts of iron dissolved, 260 parts of hydrochloric acid and 135 parts of nitric acid.

Staining.—1. Stain the section in carmin.

2. Differentiate in hydrochloric acid-alcohol.

3. Wash in alcohol.

4. Now stain the sections in Weigert's elastic-fibre stain for from twenty minutes to one hour.

5. Dehydrate in absolute alcohol.

6. Clear in pure xylol. Balsam.

By staining the nuclei with carmin first, and then staining the protoplasm with orange G later, the effect of the stain will be heightened. The elastic fibres appear dark blue or almost black. Another stain for elastic fibres is that of Unna-Taenzer.

Fat and Fatty Degeneration.—Fat is stained intensely black by osmic acid. Flemming's solution may be used for fixation. In preparations that

have been hardened in formol, the degenerative processes may be demonstrated as follows:—

1. Transfer the specimen from formol into Flemming's solution.

2. Place the sections in $^{5}/_{10}$-per-cent. strength chromic acid for three hours, then for twenty-four hours in 1-per-cent. strength chromic acid.

3. Stain in hematoxylin solution and wash in a saturated solution of picric acid. The cell-granules are blue, while the remainder is stained green (Busch).

The fat-granules within the cells have the following properties:—

1. They do not disappear when acetic acid is added.

2. They are resistant to the treatment with 1- to 3-per-cent. strength solutions of potassium and sodium hydrate.

3. They are blackened by the addition of a 1-per-cent. strength osmic acid.

4. The smallest droplets are stained intensely violet by iodin-violet.

5. They are soluble in chloroform and ether.

Fibrin.—Fibrin stains with the acid anilin dyes: picric acid, eosin, and acid fuchsin. Weigert has devised a special stain for fibrin.

Cholesterin.—Cholesterin crystals are generally found in masses of fatty *detritus*. They are frequently found in the degenerated vitreous, in subchorioidal and subretinal exudations, and in the anterior chamber. Cholesterin is soluble in alcohol and ether; hence the crystals are not visible in sections that have been obtained from specimens hardened in paraffin or in celloidin. They leave characteristic clefts or gaps if they were present. When treated with Lugol's solution (iodin, 1.0; potassium iodid, 1.0; water, 100.0), the crystals become dark brown. By adding a few drops of a 30- to 40-per-cent. strength sulphuric acid they will become gradually blue-red, blue-green, and finally blue.

Calcareous Degeneration.—Calcareous deposits occur in cells as well as in the ground-substance. The lime deposited in the tissues in the form of granules or clumps appears white and glittering when seen by reflected light; when examined by transmitted light, it appears dark. By the addition of hydrochloric acid (permitted to flow in from the edge of the cover-glass), the calcium carbonate is dissolved, with the liberation of carbon-dioxid gas, while the calcium phosphate is dissolved without. By adding sulphuric acid gypsum crystals are formed. Lime takes a characteristic blue stain with alum hematoxylin; sometimes it is reddish blue.

By Leutert's method the slightest traces of lime can be demonstrated as follows:—

1. Staining sections not imbedded in paraffin in concentrated alcoholic solution of hematein (for fifteen minutes).

2. Wash in flowing water for fifteen minutes.

3. Stain further in a 1-per-cent. strength solution of safranin (for from five to eight hours).

4. Rinse in water.

5. Differentiate and dehydrate in alcohol, oil, and balsam.

The lime is stained deep steel-blue and the nuclei intensely red. The sections are not permanent.

Mucoid Degeneration.—There are some cells in the normal conjunctiva which contain mucin. In inflammatory processes of this membrane, and on the surface of papillomata, we find them in great numbers. If the specimen has been stained with carmin, the mucin in the cell-body will remain unstained and the goblet-cells appear as light spaces in the epithelium. The nucleus at the bottom of the cell only is stained red. The mucin also is not stained by dilute solutions of hematoxylin, but it stains diffusely blue with the concentrated solutions of hematoxylin; so that the nuclei are difficult to discern.

Acid fuchsin stains the mucin intensely red, safranin stains the mucin orange-red, and methylene blue stains the mucin blue. The following is a very good method:—

1. Fix in a concentrated aqueous solution of corrosive sublimate for from two to eight hours.

2. Rapidly harden and dehydrate in absolute alcohol.

3. Imbed in celloidin or, preferably, in paraffin.

4. Place in a concentrated aqueous solution of corrosive sublimate for half a minute the sections from which the paraffin has been removed.

5. Rinse in alcohol.

6. Stain in a dilute solution of thionin (2 drops of a saturated solution of thionin to 5 cubic centimetres of water).

7. Rinse in alcohol, 90-per-cent. strength.

8. Rapidly dehydrate in absolute alcohol.

9. Clear the section in a mixture of oil of cloves, 1 part, and oil of thyme, 5 parts.

10. Oil of cedar. Balsam.

Other and more permanent stains are mucicarmin and the mucihematein of P. Meyer.

Hyalin and Colloid Degeneration.—Hyalin and colloid substances are characterized by their high refractive power. They are comparatively insoluble albuminous substances which occur very frequently in the various structures of the eye. They are found physiologically in advanced age and also in chronic inflammatory processes. For fixation formol-Müller fluid or alcohol may be used. The substances stain well with the acid anilin dyes (eosin, acid fuchsin, and picric acid). With the Van Gieson stain these bodies are colored a brilliant red. Bismarck brown gives them a light-brown tint. As a nuclear stain hematoxylin may be used.

Amyloid Degeneration.—Amyloid is very resistant to alkalies, acids, etc. Formol, sublimate, alcohol, and Müller's fluid may be used to fix. Hematoxylin, eosin, or the Van Gieson-Ernst stains are good. With the former the amyloid substance stains pink; with the latter, pink to red-brown. Besides these there are several specific stains, viz.:—

1. IODIN REACTION.—The sections are placed for from three to ten minutes in Lugol's solution diluted with 3 parts of water. The sections are then washed and examined in glycerin. The degenerated areas are stained brownish red, while the unaffected tissue is yellow. The brownish-red color is rendered still more brilliant if 25 per cent. of glycerin is added to the solution. This stain is not permanent.

2. IODIN AND SULPHURIC-ACID REACTION.—If a section has been treated as mentioned above, and is then placed in a 1-per-cent. strength solution of sulphuric acid, the brown color will become more saturated, or become violet, blue, or greenish. Sometimes some of these colors can be seen with the iodin treatment only.

3. METHYL-VIOLET AND GENTIAN-VIOLET REACTIONS.—This reaction is not absolutely reliable, as mucus may be also stained with this method. The procedure is as follows:—

1. Stain for from one-half to fifteen minutes in a 2-per-cent. strength solution of methyl-violet or in a gentian-violet solution.

2. Wash in a 2-per-cent. strength solution of acetic acid or in a 1-per-cent. strength solution of hydrochloric acid for from two to three minutes.

3. Rinse thoroughly in water.

4. Imbed in glycerin, in a concentrated solution of potassium acetate, or in levulose. The cover-glass should be rimmed if the section is to be permanent.

The amyloid areas become purple-red; the remainder of the tissue takes a blue tint.

4. THIONIN STAIN.—1. Stain in a concentrated aqueous solution of thionin for five minutes.

2. Wash in distilled water.

3. Dry on the slide with blotting-paper.

4. Dehydrate and clear with anilin oil-xylol (2 to 1).

5. Pure xylol. Canada balsam. Amyloid stains bright blue or lilac; the unaffected areas stain bluish or violet.

Corpora Amylacea.—This is a local condition which manifests itself in foci of degeneration or amyloid concretions, having a spheric outline, and are fifteen to twenty-five micromillimetres in diameter. They are found in the prostate, the brain, the optic nerve, the chiasma, and the optic tract. Sometimes they are found as far back as the corpora geniculata externa and the optic thalami. The amyloid concretions are most frequently found in ascending atrophy after phthisis bulbi. They usually give the amyloid reactions with iodin and acids. When treated with Lugol's solution, they appear brown or violet; the surrounding tissue is yellow. When stained with Lugol's solution and treated with an acid, they become violet. When these concretions are found in old corneal scars, the specimen has been fixed in Müller's fluid, and the section is stained with hemalum hematoxylin-alum-carmin, they appear yellow. In the optic nerve they stain blue with hematoxylin and hemalaun, and red with alum-carmin.

Glycogen.—The demonstration of glycogen is of little value to the ophthalmologist. The method of Langhans will be mentioned here, which is also of value in staining amyloid, and is as follows:—

1. Stain in Lugol's solution (for from five to fifteen minutes).

2. Dehydrate in a solution composed of tincture of iodin, 1.0; absolute alcohol, 4.0.

3. Clear and preserve the specimen in origanum-oil by rimming the cover.

THE DEMONSTRATION OF FOREIGN SUBSTANCES IN THE EYE.

Iron.—If particles of iron remain in the eye for long periods of time they lead to changes known as siderosis bulbi. Von Hippel distinguishes two kinds of siderosis, viz.: xenogenous siderosis, due to the presence of a foreign body which is of iron or of steel; and hematogenous siderosis, following hemorrhages and due to the iron (hemosiderin) which is usually present in the blood.

The two following methods are of service for the demonstration of iron in the eye. As a matter of course, no iron or steel teasing needles can be used in staining with these methods.

PERL'S REACTION.—The eyeball should have been hardened in formol or alcohol. Von Hippel claims the reaction may be obtained with specimens which have lain for years in Müller's fluid, although it appears more slowly.

1. Place the sections in a 2-per-cent. strength aqueous solution of ferrocyanid of potassium for a few minutes.

2. Then place them in a $^{5}/_{10}$- to 1-per-cent. strength solution of hydrochloric acid.

3. Wash the sections in water.

4. Clear the specimen and mount in balsam.

If a nuclear stain is also desired, the following staining method may be employed:—

1. Stain the sections in lithia-carmin for from one to two hours.

2. Wash in water.

3. Stain in a 2-per-cent. strength solution of ferrocyanid of potassium for from four to six hours.

4. Allow the section to remain in a 1-per-cent. strength solution of hydrochloric acid for from six to twelve hours.

5. Rapidly wash in water.

6. Dehydrate in alcohol. Origanum-oil. Balsam.

The pigment containing iron stains intensely blue.

QUINCKE'S REACTION.—1. Place the sections in a freshly prepared solution of ammonium sulphid for from ten to twenty minutes—until they have acquired a dark-green color.

2. Rinse in water.

3. Alcohol.

4. Oil. Balsam.

Should a nuclear stain be desired, the sections may be stained with alum carmin before or after placing the sections in ammonium sulphid. The iron appears in the form of dark-green granules.

Copper.—To demonstrate the presence of copper treat the sections with ferrocyanid of potassium as in Perl's reaction. Then place the section in a 1-per-cent. strength solution of acetic acid. Tissues containing copper will become brown-red, owing to the precipitation of ferrocyanid of copper.

STAINING FOR BACTERIA IN SECTIONS.

The sections should be as thin as possible in order to obtain satisfactory results. If Müller's fluid was employed for fixation, the sections should be placed in a 5-per-cent. strength solution of oxalic acid for several hours, when a satisfactory bacterial stain may be occasionally accomplished. But, in all instances where bacteria are to be stained in sections, other fixing agents, such as absolute alcohol, formol, etc., should be used.

(A) Methylene Blue.—1. Stain in Löffler's alkaline methylene-blue for ten minutes (concentrated alcoholic methylene-blue solution, 30.0; potassium-hydrate solution [1 to 10,000], 100.0).

2. Wash in water.

3. Differentiate in $^1/_2$-per-cent. strength acetic acid for from one to three seconds.

4. Wash in water.

5. Ninety-five-per-cent. strength alcohol. Absolute alcohol.

6. Xylol. Balsam.

(B) Gram-Weigert Method.—1. Stain with carmin, preferably lithia-carmin.

2. Wash in water.

3. Spread the section smoothly on the slide, on which it is to be firmly pressed with filter-paper.

4. Stain for three minutes in the following, a freshly prepared solution: $^1/_2$ cubic centimetre of anilin-oil is emulsified with 5 cubic centimetres of water by thoroughly shaking in a test-tube; the mixture is then filtered through a fine filter-paper previously moistened with water. To 10 parts of the above solution 1 part of a filtered saturated alcoholic solution of gentian-violet is added.

5. Treat with Lugol's solution (iodin, 1.0; potassium iodid, 2.0; water, 100.0) for two minutes.

6. Dry thoroughly with filter-paper.

7. Differentiate with anilin-xylol (2 to 1) until it is no longer tinged.

8. Remove the anilin-oil thoroughly with xylol.

9. Xylol. Balsam.

INDEX.

ABADIE, 276, 525, 580, 584, 695
Abducens nerve, 19
  paralysis of, 661
  radiations of, 51
Abduction, 109, 680
Aberration, chromatic, 69, 717
  spheric, 69, 716
Ablepharia, 158
Abnormal varieties of pupillary reaction, 97
Abrasion of the cornea, 336, 355
Abscess of the conjunctiva, 291
  of the cornea, 305, 308, 311
  of the eyelids, 162
  orbital, 630, 633
  subperiosteal, 629
Abscission of corneal staphyloma, 356
Absolute alcohol, 774
  glaucoma, 580
Accessory organs of the eye, 7
Accessory lacrimal glands, 22, 222
  examination of, 92
Accommodation, 66
  amplitude of, 68
  explanation of, 66
  far point of, 68
  loss of, in glaucoma, 577
  measurement of, 122
  near point of, 68
  nervous mechanism of, 67
  paralysis of, 669
  paresis of, 669
    in dental irritation, 669
  range of, determination of, 121, 122
  spasm of, 697
  terms used in recording, 67, 68
Accommodative asthenopia, 695
Acetate of lead, 255
Acetic acid, 262
Achorion Schoenleinii, 183
Acid fuchsin, 782
Acinotubular glands of Krause, 242
Acne rosacea, 180
Acoin, 302, 315
Acromegalia, 743
Actinomycosis, lacrimal, 224
  of conjunctiva, 291
Actual cautery in corneal ulcers, 314, 355
  in scleral wounds, 370
Acuity of vision, testing of, 118
Acute ascending paralysis, 742
  contagious conjunctivitis, 252
ADAMIUK, 259
ADAMS'S operation for ectropion, 208
ADDISON'S disease, 361
Adduction, 108
Adenoid vegetations, 272, 273
Adenoma of the chorioid, 415
  eyelids, 178
  lacrimal gland, 220, 625
  Meibomian glands, 179
Adherent leucoma, 264, 350
Adhesion of the iris to the cornea, 377
Adnexa, ocular, development of, 7
  physiology of, 83
Adrenalin chlorid, 255, 755
Advancement operations, 684, 685
Affections of the accessory lacrimal glands, 222
After-cataract. See *Post-operative cataract.*
After-images, 80
  negative, 81
  positive, 80
Age, influence of, upon glaucoma, 563
Agraphia, 739
Albinism, 170, 376, 412
Albino, fundus of—Fig. 2, Plate III
Albuminuric neuritis, 506
  neuroretinitis, 506
  retinitis, 505
Alcohol poisoning, 544
Alcoholic amblyopia, 544
Aleppo boil, 161
Alexia, cortical, 739
  subcortical, 739
ALFIERI'S method, 776
Allantiasis, 545
ALLEMAN, 350
ALLPORT, 197
Alopecia, 182
  areata, 182
ALT, 23, 338, 378, 598
ALTMANN, 785
Alum carmin, 780
Amakrine association fibres, 45
  cell, 45
Amaurosis (see also *Amblyopia*) following infantile convulsions, 550
  from blepharospasm, 321
  from quinin, 543
  glycosuric, 552
  partialis fugax, 557
  uremic, 552
Amaurotic cat's eye, 484
  family idiocy, 512
Ambidexterity, advantages of, 750
Amblyopia, 550
  astigmatic, 550
  central (see also *Retrobulbar neuritis*), with scotoma, 555
  congenital, 550, 747
    for colors, 550
  exanopsia, 551
  from alcohol, 544
  from hemorrhage, 553
  from non-use, 551
  hysteric, 526, 558
  malarial, 559, 747
  of nervous origin, 745
  pretended, 556
  reflex, 558, 747
  simulated, 151, 556, 747
  toxic, 542
Amblyoscope, 674
Amboyna button, 161
Ametropia, 692
  axial, 692
  medial, 692
  meridional, 692
AMMANN, 513
Ammonia causing conjunctivitis, 269
Amnesia, visual, 739
Amnesic color-blindness, 739
Amplitude of accommodation, 68
Amyloid degeneration, demonstration of, 788
  of the conjunctiva, 287
ANAGNOSTAKIS, 205
Anaphoria, 670
Anatomic changes in glaucomatous eyes, 567
  position of rest, 73
Anatomy, 8-61
  of the capsule of Tenon, 11
    cervical ganglia, 19
      sympathetic, 19
    check ligaments, 15
    chiasma, 47
    chorioid, 38
    ciliary body, 36
      ganglion, 17
      glands, 38
      muscle, 37
      processes, 37
    conjunctiva, 27

Anatomy of the cornea, 29
corneoscleral junction, 40
crystalline lens, 51
eye, 8-61
eyeball, 29
eyelids, 24
fovea centralis, 42, 46
fundus oculi, 56
interorbital space, 11
iridocorneal angle, 41
iris, 33
lacrimal apparatus, 22
glands, 22
ligamentum pectinatum, 40
macula lutea, 46
motor oculi nerve, 17, 50
nasal duct, 24
ocular lymph-spaces, 55
muscles, 12
nerves, 17
oculomotor nerve, 17, 50
ophthalmic artery, 20
nerve, 18
vein, 21
optic commissure, 47
disc, 57
nerve, 5, 41, 49
orbit, 8
orbital muscles, 12
patheticus nerve, 18, 51
retina, 5, 42
retinal blood-vessels, 58
sclera, 32
secondary eye-cup, 4
vitreous body, or humor, 3, 54
zonula of Zinn, 5, 54
ANDRADE, 310
ANDREÆ, 295
ANDREWS, 380
ANEL, 233
Anemia, 509
Anesthesia, general, 754
infiltration, 754
local, 754
of cornea, 316, 567, 576, 577
of retina, 526
Anesthetics, general, 754
local, 754
Aneurism, aortic, 97
of orbit, 101
of retinal vessels, 492
Angioid streaks in the retina, 518
Angioma cavernosum, 175
cavernous, of conjunctiva, 242
of eyelids, 175
of orbit, 621
Angle alpha, 69, 698
in hypermetropia, 698
beta, 69
gamma, 69
in myopia, 713
in strabismus, 113
negative, 113
positive, 113
metre. See *Metre angle.*
of deviation, 687
of incidence, 687
of squint, determination of, 672
refracting, 688
the visual, 65, 119
Aniridia, 376
Anisocoria. See *Inequality of pupils.*
Anisometropia, 730
Ankyloblepharon, 159, 190, 295
Annular conus, 711
posterior synechia, 387
staphyloma, 425
Anomalies of the chorioid, 411
ciliary body, 404
conjunctiva, 239
cornea, 303
crystalline lens, 437
eyeball, 617
eyelids, 158
iris, 374
lacrimal apparatus, 222, 223
optic nerve, 528
orbit, 616
retina, 481
sclera, 361
Anomalies of the vitreous humor, 473
Anophthalmos, 528, 617
Anterior chamber, angle of, anatomy of, 41
in glaucoma, 568
anomalies of, 396
cilia in, 396
changes of depth of, 396
of contents of, 396
foreign bodies in, 396
parasites in, 396
Anterior chorioiditis, 422, 426
Anterior polar cataract, 264, 308, 444
Anterior sclerotomy, 591
symblepharon, 293
synechia, 264, 309
Anteversion of the iris, 394
Anthrax, 162
Antimetropia, 730
ANTONELLI, 222
Antrum of Highmore, 228
diseases of, 633
Aortic insufficiency, 573
Aphakia, 439, 724
congenital, 438
glasses in, 725
iridectomy in, 400, 585
Aplasia of optic nerve, 528
Apoplexy of optic nerve, 549
Aqueduct of Fallopius, 188
of Sylvius, 67, 71, 97, 741
Aqueous humor, 562
composition of, in glaucoma, 562
Aquocapsulitis, 406
Arch, external tarsal, 28
tarsal, 28
Arcus senilis, 349
Arecolin, 134, 366, 576
Areolar chorioiditis, 423
Argamblyopia, 551
Argentamin, 251
ARGYLL ROBERTSON pupils, 97, 545, 741
Argyria, 167
Argyrol, 251, 255, 257, 259, 283
Argyrosis, 290
Aristol, 270, 290, 337
ARLT, 202, 206, 254, 260, 298, 379, 500, 607, 632
method of enucleation, 607, 608, 609
ARNOLD, 603
illustrations from, 52, 53
Arteria centralis retinæ, 6, 21
embolism of, 494
Arterial circle of iris, large, 34
small, 34
Arterial pulsation, 58
in glaucoma, 569, 573, 576
Arteriosclerosis, 490, 492, 493, 510, 511
Artery, anterior ethmoidal, 21
central retinal, 21
frontal, 21
hyaloid, 4, 473
lacrimal, 20
long ciliary, 20
muscular, 21
nasal, 21
ophthalmic, 20
palpebral, 28
posterior ciliary, 20
supra-orbital, 28
Artificial eyes, 614
Artificial ripening of cataract, 466
Aseptic flask, 247
ASHBY, 550
Aspergillar keratitis, 330
Aspergillus fumigatus, 330
glaucus, 330
Aspirin, 366
Associated movements, 74
Asteatosis, 180
Asteroid hyalitis, 475
Asthenopia, 745
accommodative, 695
muscular, 679
neurasthenic, 526
Astigmatic chart, 722
eye, appearance of vessels in, 142
form of image, 718
Astigmatism, 70, 692, 716
against the rule, 718
irregular, 723
regular, 717

Astigmatism with the rule, 718
Astigmometer, 116
Astigmometry, 115
Ataxia, ocular, 740, 741
  paraplegia, 742
Atheromatous ulcer of the cornea, 311
Atrabilin, 755
Atresia of the canaliculi, 223
  of the pupil, congenital, 3
Atropa belladonna, 132
Atrophia bulbi, 488
Atrophy of conjunctiva, 288
  of eyeball, 409, 422, 434, 436
  of lacrimal gland, 220
  of limbus, 340
  of optic nerve, 528, 545
  of retina, 503
Atropin, 132, 255, 266
  conjunctivitis, 259
Aura, visual, 740
Autograft, 216
Avulsion of eyeball, 647
AXENFELD, 256, 312, 429, 509
Axis, optic, 62, 68
Axonometer, 150
AYRES, 222

BAAS, 222
Bacillus of diphtheria, 271
  Klebs-Loeffler, 269, 271
  Koch-Weeks, 252, 253, 256
  of Hoffmann, 271
  tubercle, 172, 181, 289
  xerosis, 271, 288
Back-water theory of papillitis, 536
Bacteria, staining for, 791
BAER, 669
Bacterium coli commune, 263
BAKER, A. R., illustration from—Fig. 2, Plate XVIII
Balance of the ocular muscles, 101
Bandages, 754
BAQUIS, 638
BARCK, 359, 647
Basal lesions, 667
BASEDOW'S disease, 641, 745
BATES, 92, 448
BAUMGARTEN, 365
BECK, 338
BECKER, 559, 598, 645, 777
BEER, 221, 356, 460, 484, 641
BELLARMINOFF, 417
BELL'S palsy, 668
BENEDIKT, 537
BENSON, 94, 475, 496, 559
BERGER, 644, 741
BERGMEISTER, 583
BERLIN, 281, 603, 649
BERNAYS, 208
BERRY, 356, 475, 619
BERZELIUS, 442
BETTMAN, 467
BETTREMIEUX, 232
BICKERTON, 128, 613
BIETTI, 263
BILLROTH, 619, 621
Binocular field of vision, 77
  movements, 656
  single vision, 75
  vision, testing of, 151
Binoculus, 754
BIRCH-HIRSCHFELD, 543
BIRNBACHER, 199
BJERRUM'S method of perimetry, 125
BLACK, 295
Black cataract, 448, 449
"Black eye," 193, 194, 646
BLASIUS'S blepharoplasty, 214
Blastomycetic dermatitis, 166
BLED, 584
Blennorrhea of the lacrimal sac, 224
Blepharitis acarica, 186
  ciliaris, 185
  diagnosis of, 186
  eczematosa, 185, 187
  etiology of, 186
  hypertrophic, 186
  marginalis, 185
  pediculosa, 182
Blepharitis, prognosis of, 186
  squamosa, 185
  treatment of, 186
  trichophytica, 186
  ulcerosa, 185
Blepharoadenitis, 185
Blepharochalasis, 184
Blepharophimosis, 188
  congenital, 159
Blepharoplasty, methods of, 213
Blepharoptosis, 191
Blepharospasm, 189
Blepharotomy, 200
Blind spot, 78
Blindness, binocular, feigned, 151, 556
  during ocular movements, 75
  feigned, 556
  hysteric, 558
Blood in anterior chamber, 396
  -staining of the cornea, 339
Blue vision, 559
Bluestone, 259
BOCK, 262
BOECKMANN, 284, 301
BÖHM, illustrations from, 30, 31, 36, 37, 40, 59
Boil, Aleppo, 161
  Delhi, 161
BOLL'S retinal purple, 80
BONNET'S method of enucleation, 607, 608
Borax-carmin, 780
Boric acid, 284
BORSCH, 555
Botulism, 545
BOUCHERON, 561
BOWMAN'S layer of the cornea, 30, 246
  probe, 235
BRACHET, 300
BRADY, 223
Brain, abscess of, 535
  tumor of, 508
BRAMWELL, 508
BRETTAUER, 468
"Bridge coloboma," 374
BRIGHT'S retinitis, 505
BROCA'S lobe, 739
BROEKAERT, 207
BRONNER, 175, 437, 594
Brossage, 262, 283
BROWN-SÉQUARD, 537
BRÜCKE, 38
BRUNS, 643
BRYANT, 612
BUCHANAN, 557
BUCKLEY, 292
Bulbar paralysis, 742
BULL, C. S., 380, 388, 427, 429, 430, 511, 576, 630, 633
Bullæ of conjunctiva, 275
  of cornea, 329
  of eyelids, 165
BULLER, 222, 280
Buller's shield, 265, 270, 272
Bullous keratitis, 329
Buphthalmos, 582
BURCHARDT, 121
BURNETT, S. M., 242, 281, 364
BUTLER, 642
BUXTON, 128
BYERS, 222, 530, 531

Calcareous cataract, 445
  degeneration, demonstration of, 787
  keratitis, 334
CALDWELL'S lacrimal probes, 236
  operation on obliterated nasal canal, 237
CALLAN, 382, 466
CALMETTE'S serum, 174
Calomel, 274, 371
Campimeter, 124
Canal for the nasal duct, 10
  hyaloid, 4, 56
  of Cloquet, 54, 473
  of Schlemm, 41
    rupture of, 369
Canaliculi, anomalies of, 223
Canaliculus, actinomycosis of, 224
  foreign bodies in, 223
  leptothrix in, 223
  mucocele of the lower, 223
  polyp of, 223

Canaliculus, slitting of, 234
streptothrix of, 224
wound of, 193
Cancroid ulcer, 177
Canites, 601, 743
Cannabis Indica, amblyopia from, 542
Canthoplasty, 200
Canthotomy (see also *Blepharotomy*), 161, 162, 189
Capsular advancement, 685
cataract, 446
Capsule of Bonnet, 11
of Tenon, 11
advancement of, 685
inflammation of, 632
Capsulo-pupillary membrane, 466
Capsulotomy, 452, 460
immediate, 460
Carbol-fuchsin, 256
Carbolic acid in corneal ulcers, 314
Carbon bisulphid, amblyopia from, 542
Carbuncle, 162
Carcinoma of caruncle, 296
of chorioid, 414
of ciliary body, 405
of conjunctiva, 246
of eyelid, 176
of lacrimal gland, 221
of orbit, 621, 625
Cardinal points, 63
Care of the eyes during school-life, 762
Caries of orbit, 535, 627
CARPENTER, 388, 413
Carron-oil, 194
CARTER, 357
Caruncle, abscess of, 296
affections of, 295
anatomy of, 25
carcinoma of, 296
cylindroma of, 296
dermoid of, 296
fibroma of, 296
lithiasis of, 296
lymphangioma of, 296
sarcoma of, 296
sinking of, after tenotomy, 683
trichosis of, 296
tumors of, 296
Case history, 85
CASPAR, 518
Cassaripe, 316
Cataphoria, 670
Cataract, 439
accrete, 448
anterior polar, 264, 308, 444
artificial ripening of, 466
axial, 443
black, 448
calcareous, 445
capsular, 439, 446, 447
central, 444
complete, 443
complicated, 448
congenital, 443
coralliform, 444
course of, 442
delivery of, 453
diabetic, 449
diagnosis of, 441
etiology of, 442
extraction of, 451
in its capsule, 459
linear, 460
of immature, 461
with iridectomy, 453
with Kalt suture, 459
without iridectomy, 451
without speculum, 461
glaucomatous, 568
gray, 439
immature, extraction of, 461
immediate capsulotomy in extraction, 460
incipient, 441
incomplete, 443
juvenile, 444, 463
lactate, 445
membranous, 445
milky, 445
Morgagnian, 442
operations for, 449
Cataract, operations for, choice of, 458
results of, 468
osseous, 445
pathology of, 442
posterior polar, 444
post-operative, 446, 465
prognosis of, 443
punctate, 444
secondary, 448
senile, 439
simple, 439
soft, 444
spontaneous cure of, 468
spontaneous delivery of, 453
stellar, 444
traumatic, 445, 446, 464
treatment of, 443, 444, 445, 446
zonular, 444
Catarrh, follicular, 257
of lacrimal sac, 225
Catarrhal ulcer, 253
Caterpillar-hairs, 285
Catoptric images, 67
test, 441
Cautery. See *Actual cautery.*
Cavernous sinus, compression of, 536
thrombosis of, 636
Celloidin imbedding, 777
Cellulitis of orbit, 630
Centrad, 688
Centre of rotation, 68, 72
visual, 82
Cerebellum, tumors of, 534
Cerebral layer of the retina, 44
Cerebritis, 535
Cerebrospinal meningitis, 740
CHABANEIX, 230
Chalazion, diagnosis of, 182
etiology of, 182
external, 181
internal, 181
operations for, 195
pathology of, 181
symptoms of, 181
treatment of, 182
CHALK'S eye-drop bottle, 247
Chamber, anterior. See *Anterior chamber.*
posterior. See *Posterior chamber.*
CHANCE, 276
CHANDLER, 316
CHARCOT, ophthalmic migraine of, 191
CHARTRES, 264
CHAUFFARD, 584
Chaulmoogra-oil, 174
CHEATHAM, 284
Chemosis, 253, 256, 263, 266, 276, 293, 631, 632
Cherry-red spot in macula, 495
CHESELDEN, 400
Chiasma, optic, 47
Chloasma, 167
Chlorin-water, intra-ocular injections of, 475
Chloroform, 754
Chloroma, 222
Chlorosis, 535
Choked disc, 533, 534, 535
Cholesteatoma of iris, 378
Cholesterin, demonstration of, 787
Chondroma of lacrimal gland, 222
Chorea, 496, 744
Chorioid, adenoma of, 415
albinism of, 412
anatomy of, 38
angiosarcoma of, 415
carcinoma of, 414
central senile areolar atrophy of, 424
chronic inflammatory processes in, 486
colloid changes in, 435
coloboma of, 411
congenital anomalies of, 411
congestion of, 420
cysts of, 414
detachment of, 417, 434
diseases of, 411-436
enchondroma of, 415
foreign bodies in, 433
gumma of, 419
hemorrhage into, 433
hyperemia of, 420
inflammation of, 420
injuries of, 433

Chorioid, large tubercular mass in, 413
leucosarcoma of, 486
leucosis of, 412
macular coloboma of, 412
myoma of, 420
nevi of, 420
ossification of, 420
perithelioma of, 415
rupture of, 434
sarcoma of, 415
diagnosis, 416
metastatic, 415
pathology, 419
prognosis, 419
symptoms, 416
treatment, 419
sclerosis of vessels of, 424
senile areolar atrophy of, 424
solitary tubercle of, 418
telangiectatic sarcoma of, 415
tuberculosis of, 412, 486
tumors of, 414
wounds of, 433
Chorioidal craters, 522
hemorrhage, 433
Chorioiditis, 420
acute, 421
anterior, 422, 426
areolar, 423
atrophic, 421
causes of, 427
central, 422, 423
"central senile guttate," 424
character of pigment in, 421, 428
clinical forms of, 422
diagnosis of, 427
diffuse syphilitic, 501
disseminated, 422, 423
exudative, non-suppurative, 420
from errors of refraction, 427
gonorrheal, 422, 427
guttate, 424
hereditary, 427
metastatic, 429
myopic, 422, 425
old, 428
pathology of, 427
plastic, 420
prognosis of, 428
purulent, 429
recent, 428
serous, 420
suppurative, 429
symptoms of, 421
syphilitic, 422, 426. See *Syphilitic retinitis.*
Tay's, 424, 516
treatment of, 428
with descemetitis, 422, 426
Chorioidoretinitis, 420, 559
striata, 518
sympathetic, 600
syphilitic, 426, 501, 504, 559
Chromatic aberration, 69, 717
Chromidrosis, 179
Chronic catarrhal conjunctivitis, 256
Chronic conjunctivitis, 277
Cicatricial orbit, operations for, 615
pterygium, 287
Cilia. See *Eyelashes.*
Ciliary body, anatomy of, 36
angiomata of, 405
calcification of, 405
carcinomata of, 405
coloboma of, 404
cysts of, 405
diseases of, 404
fibromyomata of, 405
foreign bodies in, 409
gumma of, 405
inflammation of, 406
plastic, 408
purulent, 409
injuries of, 409
myomata of, 405
ossification of, 405
sarcomata of, 405
tuberculosis of, 405
tumors of, 404
wounds of, 409
congestion, 248
Ciliary ganglion, 17
glands, 38, 407
injection, 248
muscle, 37
part of the retina, 46
processes, 37
ring, 36
staphyloma, 366
vessels, anterior, 91, 248, 580
Cilioretinal artery, 59
vein, 59
vessels in embolism, 496
Cinchona, amblyopia from, 543
Cinnamate of sodium, 302
CIRINCIONE, 225, 532
Citrate of copper, 283
Clearness of vision, 81
Climatic changes in their effect on the eyes, 770
CLOQUET, canal of, 54, 473, 478
Cnidosis, 160
Cobalt-glass test, 677
Cocain, 255, 451, 754
COE, 297
Coffee, amblyopia from, 542
COHN, 553, 707
COHNHEIM, 740
COLLINS, TREACHER, 51, 156, 305, 339, 340, 374, 407, 437, 458, 496, 578, 583
COLLINS, W. J., 442
Colloid bodies in nerve-head, 532
degeneration of the skin, 179
disease of the macular region, 435
Collyrium, application of, 250
Coloboma, macular, 412
of chorioid and retina, 411
of cornea, 304
of eyelid, 158
of iris, 374
of lens, 437
of retina, 411, 481
of sheath of optic nerve, 528
of vitreous humor, 473
Colon bacillus, 263, 269
Color-field of vision, 78
reversal of, 558
Color-perception, theories of, 83, 550
variation of normal, 83
Color-sense, testing of, 127
Comitant convergent squint, 671
divergent strabismus, 671
Commotio retinæ, 521
Concave lens, definition of, 688
retinoscopy mirror, 147
Concussion of the retina, 521
Congenital cataract, 443
cupping of the optic disc, 529
hyperkeratosis, 167
iritis, 388
luxation of the lens, 439
paralysis, 733
ptosis, 159, 191, 196
Congestion of posterior conjunctival vessels, 248
Conic cornea, 344
operative treatment of, 355
optic treatment of, 345
Conium, 190
Conjugate foci, 689
lateral deviation, 744
paralysis, 733
Conjunctiva, abscess of, 291
actinomycosis of, 291
acute blennorrhea of, 262
amyloid degeneration of, 287
anatomy of, 27
angioma of, 242
argyrosis of, 290
atrophy of, 288
blood-vessels of, 90, 248
burns of, 294
calcification of, 284
carcinoma of, 246
cavernous angioma of, 242
chancre of, 291, 292; Fig. 7, Plate VII
chemosis of, 293
congenital anomalies of, 239
copper-colored spots in, 292
cylindroma of, 246
cysticercus of, 243

Conjunctiva, cysts of, 242
dermoid tumors of, 240
diseases of, 239-302
dry catarrh of, 248
ecchymosis of, 293
edema of, 253
emphysema of, 293
epibulbar sarcoma of, 244
epithelioma of, 245, 246
essential shrinking of, 165, 193, 275
examination of, 89
fibroma of, 240
fibro-osteomata of, 242
folliculosis of, 257
foreign bodies in, 294
granulation tumors of, 241
grattage of, 301
gumma of, 292
hemorrhage beneath, 293
hemorrhage from, 291
herpes of, 274
hyperemia of, 248
symptoms, 249
treatment, 249
inflammations of, 247
injuries to, 294
leprosy of, 290
leucosarcoma of, 244
lime-burns of, 294
lipoma of, 240
lithiasis of, 276
lupus of, 291
lymphangiectasis of, 243
lymphoma of, 275
melanosarcoma of, 244
mucous patches of, 292
myxoma of, 241
necrosis of, after injections, 302
operations on, 296
osteoma of, 242
papillomata of, 241
pemphigus of, 275
phlebolith of, 242
pigmentation of, in trachoma, 280
polypi of, 241
sarcoma of, 244
scalds of, 294
siderosis of, 290
spring catarrh of, 260
symblepharon of, 293
syphilis of, 291
telangiectases of, 171
tuberculosis of, 289
tumors of, 239
ulcers of, 292
vaccinia of, 284
varicella of, 284
vascular tumors of, 242
wounds of, 295
xerosis of, 288
Conjunctival injection, 247, 248
sac, anatomy of, 25
temperature of, 239
vessels, anterior, 91
posterior, 90
Conjunctivitides, 250
Conjunctivitis, 247
acute contagious, 252
clinical varieties of, 254
diagnosis of, 253
etiology of, 252
pathology of, 252
prognosis of, 254
symptoms of, 253
treatment of, 254
atropin, 259
catarrhal, 251
caterpillar-hair, 285
chronic, 277
catarrhal, 256
etiology, 257
treatment, 257
croupous, 269
diagnosis, 270
etiology, 269
symptoms, 270
treatment, 270
diphtheritic, 271
diagnosis, 271
etiology, 271
Conjunctivitis, diphtheritic, symptoms, 271
treatment, 272
diplobacillus, 256
eczematosa, 272
Egyptian, 277
exanthematous, 254
follicular, 254, 257
diagnosis, 258
etiology, 258
symptoms, 258
treatment, 258
from electric light, 254
from x-rays, 254
gonorrheal, 262
of the adult, 263
complications, 264
diagnosis, 264
etiology, 264
prognosis, 265
prophylaxis, 265
symptoms, 263
treatment, 265
of the newborn, 267
diagnosis, 267
etiology, 267
prognosis, 267
prophylaxis, 267
symptoms, 267
treatment, 268
granular, 277
complications, 280
diagnosis, 281
etiology, 277
forms, 278
pathology, 281
prognosis, 282
treatment, 282
Koch-Weeks bacillus, 252
lacrimal, 277
membranous, 269
metastatic gonorrheal, 254
military, 277
muco-purulent, 252
neonatorum. See *Conjunctivitis, gonorrheal.*
papular, 275
Parinaud's, 275
petrificans, 284
phlyctenular, 272
diagnosis, 273
etiology, 272
prognosis, 273
symptoms, 273
treatment, 274
plastic, 269
pneumococcic, 256
pseudomembranous, 269
purulent gonorrheal, 262
non-gonorrheal, 268
treatment, 269
pustular, 254
simple, 251
spring, 260
diagnosis, 260
etiology, 260
pathology, 261
prognosis, 261
symptoms, 260
treatment, 261
subacute catarrhal, 256
toxic, 259
traumatic, 254
uratic, 276
variolar, 276
vernal, 260
Construction of school-buildings, 763
Contraction of the pupil, 71
Conus, 367, 425, 528, 710
Convergence, amplitude of, 124
and accommodation, 123
excess in esophoria, 679
excessive, 679
far point of, 123
insufficiency of, 679
measurement of, 123
near point of, 123 678
negative, 123
positive, 124
power of, how measured, 678
reflex of pupil. See *Robertson symptom.*
test of, 124

Convex lens, virtual image of, 64
COOVER, 295
Copper, demonstration of, 791
COPPEZ, 269, 271, 312
Corectopia, 375
Corelysis, 393, 397
Cornea, abrasion of the, 355
abscess of, 306, 308, 311
anatomy of, 29
anterior elastic lamina of, 30
atheromatous ulcer of, 311
atrophy of limbus of, 340
blood-staining of, 339
blood-vessels of, 31
in disease, 306
Bowman's layer of, 30
canaliculi of, 30
causes of infective ulcers of, 309
cauterization of, 355
cells of, 30
changes in, in exophthalmic goitre, 644
classification of inflammations of, 307
collapse of, 453
congenital anomalies of, 303
conic, 344
corpuscles of, 30
creeping ulcer of, 311
crescentic ulcer of, 311
cyst of, 305
dendritic ulcer of, 311, 331
dermoid of, 304
diameter of, normal, 29
discoloration of, with blood-pigment, 339
diseases of, 303-360
ectasia of, congenital, 304
eczema of, 320
edema of, in glaucoma, 567
endothelium of, 31
epithelioma of, 305
examination of, 92, 93
excision of, 356
exhaustion ulcer of, 318
fibrillæ of, 30
fibroma of, 305
fistula of, 346
fistulous staphyloma of, 346
foreign bodies in, 354
fungus infection of, 309, 330
globosa, 345
healing of wounds of, 353
hemorrhage into, 353
herpes of, 336
after cataract extraction, 456
horizontal meridian of, 29
infantile ulceration of, with xerosis of conjunctiva, 318
injuries of, 351
inspection of, 92
keloid of, 305
keratocele, 308, 346
lepra of, 341
mechanical injuries of, 352
onyx, 307
opacities of, 349
operations on, 355
paracentesis of, 355
pemphigus of, 329
perforation of, 308, 314, 315
posterior elastic lamina of, 31
protrusions of, 341
relapsing herpes of, 333
ribbon-shaped opacity of, 334
ring ulcer of, 310
rodent ulcer of, 310
round central ulcer of, 310
Saemisch's incision of, 315, 357
sclerosis of limbus of, 340
subepithelial opacity of, 335
siderosis of, 341
staphyloma of, 341
tattooing of, 359
tonsure of, 301
transverse film of, 334
tuberculosis of, 341
xanthelasma of, 341
zonular opacity of, 334
Corneal ulcers in trachoma, 280
wounds, healing of, 353
Corneitis. See *Keratitis*.
Cornu cutaneum, 168
CORNWALL, 349
Corpora amylacea, demonstration of, 789
geniculata, 41
quadrigemina, 41
Corresponding points of the retina, 75
Cortical reflex of Haab, 98
"Couching" of cataract, 462
Cover test for heterophoria, 677
CREDÉ'S method, 268
Creeping ulcer of the cornea, 311
Creolin, 260
"CRICK'S" dots, 515
CRITCHETT, 242, 343, 531, 605
operation for staphyloma of cornea, 356
Croupous conjunctivitis, 269
Cryptophthalmos, 158
Crystalline lens, absence of, 439
anatomy of, 51
calcification of, 445
capsule of, 54
changes in, in accommodation, 66
coloboma of, 437
congenital anomalies of, 437
congenital luxation of, 439
dimensions of, 51
diseases of, 437-472
dislocation of, 439, 469
examination of, 98
fetal, 3
foreign bodies in, 472
growth of, 51
increase in size of, 51
injuries of, 471
luxation of, 439, 469
opacity of, 439
operations upon, 449
ossification of, 471
parasites in, 471
regeneration of, 471
removal of, in myopia, 716
spontaneous delivery of, 453
spontaneous extrusion of, 471
structure of, 52
suspensory ligament of, 54
swelling of, 440, 464
Cuneus, 41
Cuprol, 255
Curettement, 162
Cutting of sections, 779
the eyeball, 777
Cyanopsia, 559
Cyanuret of mercury, 302
Cyclicotomy, 590
Cyclitis, 406, 408
after cataract extraction, 456
pathology, 407, 409
plastic, 408
prognosis of, 407
purulent, 409
serous, 406
symptoms of, 407, 408
traumatic, 409
treatment of, 407, 409
varieties, 406
Cyclophoria, 101
test for, 677
Cyclopia, 616
Cycloplegia, 131, 669
Cycloplegics, 132
Cylindric lenses, 690
Cylindroma of conjunctiva, 246
Cyst, dermoid, of conjunctiva, 240
gelatinous of conjunctiva, 243
of chorioid, 414
of cornea, 305
of eyelid, 167
of iris, 377
of orbit, 618, 625
of retina, 482
of sclera, 361
sebaceous, of eyelid, 180
subconjunctival, 242
Cysticerci of conjunctiva, 243
of eyelids, 183
of iris, 381
of retina, 488
of vitreous humor, 478
Cysticercus, intra-ocular, 418
Cystoid cicatrix, after cataract extraction, 457
Cystotomy, 452, 462

CZERMAK, 192, 357, 606

Dacryoadenalgia, 220
Dacryoadenitis, acute, 219
  chronic, 220
Dacryocystitis, catarrhal, 225
  suppurative, 226
Dacryolith, 220
Dacryops, 221
Dactylitis, 272
DALRYMPLE'S sign, 87, 644
Daltonism, 550
DANVERS, 246, 261, 262
DARIER, 262, 315, 321, 339, 362, 363, 391
Darier's disease, 172
DAVIDSON, 154, 651
DAVIS, A. E., 114
Day-blindness, 557
DE BECK, 173, 351, 375, 395
DE LAPERSONNE, 433
DE SCHWEINITZ, 260, 415, 435, 487, 519, 639, 640
DE WECKER, 284, 343, 349, 357, 359, 400, 401, 403, 513, 592
  operation for staphyloma, 357
  scissors of, 399
    in secondary cataract operations, 466
Decalcification of eye specimens, 775
Defects in the visual field, 126
Dehydration of sections, 784
DEJERINE, 739
DELAFIELD'S hematoxylin, 780
Delhi boil, 161
Delirium after cataract extraction, 454
DEMICHERI, 291
Demodex folliculorum, 186
DEMOURS, membrane of, 31
Dendritic keratitis, 331
Dendritic ulcer of the cornea, 311, 331
DENIG, 638
DENNETT'S method of measuring prisms, 683
DÉPOUTOL, 647
Depression of cataract, 462
Depth, sense of, 76
DERBY, 179, 548, 707
Dermatitis, 165, 457
  blastomycetic, 166
  herpetiformis, 165
  medicamentosa, 165
  venenata, 165
Dermoid cyst of eyelid, 167
  of orbit, 618, 619
  of caruncle, 296
  of conjunctiva, 239, 240
  of cornea, 304
Dermolipomata, 240
DESCEMET, membrane of, 31
Descemetitis, 333, 406
Descending neuritis, 534
DESCHAMPS, 400
  iridectomy in aphakic eyes, 400
DESMARRES, 202, 230, 392
  depletion, 392
DESPAGNET, 275, 300
Detachment of chorioid, 417
  of retina, 416, 522
    etiology of, 522
    in myopia, 710
    pathology of, 523
    prognosis of, 524
    symptoms of, 523
    treatment of, 524
  of vitreous, 480, 710
DEUTSCHMANN, 594, 600
Development of the eye, 1
Diabetic cataract, 449
  coma, 449
  iritis, 389
  retinitis, 503
Diameter of cornea, 29
  of pupil, 71
Dichromasia, 550
DIEFFENBACH'S operation for ectropion, 208
Diffuse dots in the retina, 516
Dilation of the pupil, 71
Dilator pupillæ, 34
Diminishing hypermetropia, 692
DIMMER, 436, 493
Dionin, 321, 391, 392
Dioptre, the, 688
Dioptric apparatus, 62
Diphtheria antitoxin, 179, 270, 272
Diphtheritic conjunctivitis, 271
Diplobacillus conjunctivitis, 256
  of Morax and Axenfeld, 256
Diplococcus intracellularis meningitidis, 269
Diplopia, 72, 661, 662, 663
  crossed, 663
  horizontal, 661
  in the lower field, 662
  in the upper field, 662
  monocular, 72
  turning of head to avoid, 659
  vertical, 662
Direct method of ophthalmoscopy, 136, 137
Direct vision, 79
Disc, optic, 57
Discission of cataract, 448, 463
  glaucoma after, 448
  of post-operative cataract, 465
Disc-like keratitis, 339
Dislocation of eyeball, 647
  of lacrimal gland, 221
  of lens, acquired, 469
    diagnosis of, 469
    prognosis of, 470
    treatment of, 470
  congenital, 439
  intentional, 462
Disseminated sclerosis, 740
Distichiasis, 190
  congenital, 159
Divergence excess, 679
  insufficiency, 679
DIXON, 43
DOBELL'S solution, 228
DOCK, 222
DODD, 504, 559
DODGE, 75
DOJER, 72
DOMINIQUE, 275
DONDERS, 58, 72, 562, 726
DOR, 525
Double vision. See *Diplopia*.
DOUGLASS, 185
DOYNE, 437
DRANSART, 199
Dressings, preparation of, 753
Dropping of fluid from the nostril, 541
DRUAULT, 312
Drug conjunctivitis, 259
Drusenbildungen in optic papilla, 532
DUANE, 663, 664, 676, 678
Duboisin, 133
DUCHENNE'S disease, 188
Duct, lacrimo-nasal, 24
  obstruction of, 227
  probing of, 235
  stricture of, 227
DUDLEY, 559
DUNN, 439, 463
Duration of visual sensations, 80
DYER, L., 174
Dyschromatopsia. See *Dichromasia*.
Dyslexia, 739

Ear disease, 637
Ecchymosis of conjunctiva, 293
  of eyelids, 193
  subconjunctival, 293
Echinococcus in orbit, 626
  subretinal, 489
Ectasia of cornea, 304
  scleræ, 366
Ectoderm, 2
Ectogenous panophthalmitis, 430
Ectopia lentis, 439
Ectropion, 192, 223
  acute, 207
  cicatricial, 192
  congenital, 159
  of uvea, 376
  operations for, 207
  organic, 280
  spastic, 280
  treatment of, 192
  varieties of, 192
Eczema, 160
  neurotic, 160
  of conjunctiva, 272
  of cornea, 320

Eczema of eyelids, 160
of nose and lip, 273
seborrheic, 160
strumous, 160
Edema of conjunctiva, 253
of cornea, 567
of eyelids, 168, 169
Edematous swelling of the retina, 521
EDMUNDS, 538, 553
EDSALL, 546
Egg-film, 295
Egyptian ophthalmia, 277
Elastic fibres, demonstration of, 786
Electric ophthalmia, 514
Electricity in herpes zoster ophthalmicus, 164
in lacrimal stricture, 230, 238
in opacities of the cornea, 350
in paralytic strabismus, 668
Electrolysis in angiomas, 175
in detachment of the retina, 523
in fistula of the cornea, 349
in trichiasis, 190, 201
in granular conjunctivitis, 283
in keloid of eyelid, 174
in lacrimal diseases, 230, 238
in molluscum epitheliale, 172
in nævus vasculosus, 175
in xanthoma, 172
Electromagnet, giant, 371
of Haab, 371
of Hirschberg, 371
of Johnson, 371
of Lippincott, 371
of Sweet, 371
results of use of, 372
Elephantiasis Arabum of eyelids, sporadic type, 169
tropical type, 169
ELLETT, 350
ELSCHNIG, 224, 496
Embolism of central retinal artery, 494
Embryotoxon, 303
Emmetropia, 691
ophthalmoscopy in, 142
retinal image in, 139, 691
skiascopy in, 147, 148
Emphysema of conjunctiva, 293
of eyelids, 184
Empyema of antrum, 634
of frontal sinus, 634
of sphenoidal sinus, 634
Encanthis, 296
Encephalocele, 618
Encephalopathy, lead, 508
Enchondroma of orbit, 623
Endarteritis, 490, 496
Endocarditis, 496
Endogenous panophthalmitis, 429
Endothelioma of orbit, 621, 625
Enophthalmos, 101, 589, 640, 649
Entropion, 193, 223, 271, 280, 294, 295
congenital, 159
from trachoma, 193, 280
operations for, 203
organic, 280
senile, 193
spasmodic, 193
Enucleation of eyeball, 606, 607
accidents during or after, 609
Arlt's method, 608
Bonnet's method, 608
indications for, 606
Eosin, 782
Ephidrosis. See *Hyperidrosis*.
Epibulbar tumors, 244, 246
Epicanthus, 159
operations, 206
Epidemic ophthalmia, 252
Epilepsy, 679, 744
Epileptic seizure in cataract extraction, 454
Epiphora, 222, 226, 227
Episcleral tissue, 33
Episcleritis, 361
fugacious, 363
partialis fugax, 363
Epithelial xerosis, 288
Epithelioma of conjunctiva, 246
of cornea, 304
of eyelids, 176
Equator, 72
Equilibrium test for heterophoria, 676
Ergot, 580
amblyopia from, 542
ERISMANN, 707
ERLITZKI'S fluid, 774
Errors of refraction, 686
Erysipelas of eyelids, 163
Erythropsia, 476, 556, 559, 748
after cataract operation, 559
Eserin, 267, 576
after cataract extraction, 454
and cocain in glaucoma, 576
in glaucoma, 574
Esophoria, 670
Esotropia, 670, 671
Essential phthisis bulbi, 436
Essential shrinking of conjunctiva, 165, 193, 275
Ether, 754
Ethmoidal sinuses, diseases of, 633
Ethyl bromid, 754
Eucain, 754
EVERSBUSCH, 196, 199
Eversion of eyelids, 88
EVETSKY, 224, 419
Evisceration of the eye, 610
Hall's method, 611
Mules's method, 611
Eviscero-neurotomy, 612
EWING, 121, 206, 306
Examination of the eye, 85-157
anterior chamber, 94
caruncle, 92
children's eyes, 88
ciliary body, 99
color-sense, 127
conjunctiva, 89
cornea, 92
crystalline lens, 98
eyelashes, 86
eyelids, 87
field of vision, 124
fundus oculi, 134, 140
iris, 95
lacrimal apparatus, 91
lens, 98
light-sense, 127
macula lutea, 139
muscle-balance, 101
muscle-power, 108
ophthalmoscopic, 135
optic nerve, 139
orbit, 100
pupil, 95
pupillary area, 98
refraction, 115, 132, 141, 144-151
sclerotic, 92
visual acuity, 118
visual field, 124
vitreous humor, 99
Excavation of optic-nerve head, 570
atrophic, 570
glaucomatous, 570
physiologic, 570
Excision of cul-de-sac, 283, 299
of eyeball. See *Enucleation*.
of lacrimal sac, 234
of superior sympathetic ganglion, 589
Exciter, 594
Exciting eye, 594
Exenteration of the orbit, 653
Exfoliation of the iris, 376
Exophoria, 670
Exophthalmic goitre, 641, 745
eye symptoms of, 745
nature of, 645
prognosis of, 645
symptoms of, 641
treatment of, 645
Exophthalmos, 100, 271, 649
intermittent, 640
pulsating, 638
Exothyropexy, 645
Exotropia, 670, 671
Expression, 283
operation in trachoma, 283
External ophthalmoplegia, 665
External rectus muscle, 14
paralysis of, 661
Extirpation of lacrimal sac, 234
Extraction of intra-ocular cysticercus, 478

Extraction of immature cataract, 461
of senile cataract, 451
Exudative chorioiditis, 420, 422
Eye, anatomy of, 8-61
cardinal points of, 63
nodal point of, 62, 65, 66
optical defects of, 68
-shade, 761
Eyeball, anatomy of, 29
atrophy of, 436
dislocation of, 647
foreign bodies in, 370
spontaneous rupture of, 418
Eyelids, abscess of, 162, 194
absence of, 158
acne rosacea of, 180
Adams's operation on, 208
adenoma of Meibomian gland of, 178
albinism of, 170
Aleppo boil of, 161
alopecia areata of, 182
alopecia of, 182
Amboyna button of, 161
anatomy of, 24
ankyloblepharon, 159, 190
anomalies of, 158
anthrax of, 162
argyria of, 167
arteries of, 28
asteatosis of, 180
Biskra button of, 161
Blasius's operation on, 214
blastomycetic dermatitis of, 166
blepharitis acarica, 186
marginalis, 185
squamosa, 185
ulcerosa, 185
blepharochalasis, 184
blepharophimosis, 159, 188
congenital, 159
blepharospasm, 189
bullous affections of, 165
burns of, 194
cancroid ulcer of, 177
carbuncle of, 162
carcinoma of, 176
chalazion, 181
operations, 195
chancre of, 173
chloasma of, 167
chromidrosis of, 179
colloid degeneration of, 179
coloboma of, 158, 192, 193
congenital anomalies of, 158
hyperkeratosis of, 167
ptosis of, 159
contusion of, 193
cornu cutaneum of, 168
cryptophthalmos, 158
cyst of, 167
cysticercus of, 183
Darier's disease of, 172
Delhi boil of, 161
dermatitis of, 165
blastomycetic, of, 166
medicamentosa, of, 165
dermoid cyst of, 167
Dieffenbach's operation on, 208
discoloration of, 167
distichiasis of, 159, 190
drooping of, 191
ectropion of, 192, 207
congenital, 159
eczema of, 160
edema of, 168, 567
solid, 169
elephantiasis of, 169
emphysema of, 184
entropion of, 193
congenital, 159
epicanthus, 159
operations, 206
epithelioma of, 176
erysipelas of, 163
erythema of, 160
Ewing's operation on lower lid, 206
examination of, 87
favus of, 183
fibroma of, 175
fissures of, 161
Eyelids, fistula of, 159
foreign bodies in, 195
frambesia of, 161
freckles of, 170
Fricke's operation on, 214
Fuchs's operation on, 200
furuncle of, 161
furunculus orientalis of, 161
gangrene of, 179
gumma of, 173
gunpowder-grains in, 167, 195
hailstone of, 181
hare's eye, 187
Harlan's operation on the upper lid, 205
Hasner d'Artha's operation on, 214
hernia of fatty tissue of, 168
herpes of, 163
hives of, 160
hordeolum of, 166
Hotz's operations on, 204, 210
hyperemia of margins of, 185
hyperidrosis of, 179
hyperkeratosis of, 167
hysteric alopecia of, 182
impetigo of, 161
in glaucoma, 567
injuries of, 193
intermarginal line of, 26
Jacob's ulcer of, 177
Jaesche-Arlt operation on, 202
Kaposi's disease of, 171
keloid of, 174
keratosis follicularis of, 172
Knapp's operation on, 215
Kuhnt's operation on, 210
lacerations of, 193
lagophthalmos, 187
lentigo of, 170
leprosy of, 174
lichen of, 165
tropicus of, 180
lipoma of, 168
lupus erythematosus of, 172
vulgaris of, 173
lymphangioma of, 175
lymphatics of, 28
lymphoma of, 175
malignant pustule of, 162
marginal blepharitis, 185
measles of, 184
Meibomian cyst of, 181
microblepharon, 159
miliaria of, 180
milium of, 180
molluscum contagiosum of, 172
epitheliale of, 172
Mules's operation for ptosis of, 198
Müller's operation on, 210
muscles of, 26
nævus pigmentosus of, 170
vasculosus of, 175
nerves of, 28
neurofibroma of, 175
noma of, 179
operations on the, 195
Panas's operation on upper lid, 197
parasitic skin diseases of, 182
pemphigus of, 165
perifolliculitis of, 166
phagedenic ulceration of, 179
phosphoridrosis of, 179
phtheirinsis of, 186
pian of, 161
plexiform neuroma of, 175
"prickly heat" of, 180
psorophthalmia, 185
ptosis of, 159, 191
punctured wounds of, 193
rhagades of, 161
Robertson's operations on the, 208, 209
rodent ulcer of, 176, 177
rubeola of, 184
Sanson's operation on, 208
sarcoma of, 177
scalds of, 194
scarlatina of, 184
seborrhea of, 180
of palpebral margins, 185
serpiginous syphilide of, 173
siderosis of, 167

Eyelids, skin diseases of, 160
Snellen's operations on, 203, 207
spontaneous gangrene of, 179
steatoma of, 180
structure of, 25
stye, 166
sudamina of, 179
sycosis tarsi, 185
symblepharon, 190
syphilis of, 173
Tansley's operation on upper lid, 197
tarsal tumor, 181
tarsitis, 162
tarsorrhaphy, 200
tattooing of, 167
tinea tarsi, 185
trichiasis, 190
operations for, 201
tuberculosis of, 172
ulcer, Jacob's, 177
uridrosis of, 179
urticaria of, 160
vaccinia of, 184
varicella of, 184
varicose veins of, 170
variola of, 183
veins of, 28
verruca of, 168
vitiligo of, 170
von Ammon's operations on, 201, 208
von Graefe's operation on, 196, 200
von Langenbeck's operation on, 208
Wicherkiewicz's operation on, 207
Wilder's operation on upper lid, 196
wounds of, 193
xanthoma of, 172
xeroderma pigmentosum, 171
yaws of, 161
EYRE, 289

Facial palsy, 668
Facultative hypermetropia, 697
FALCONE, 292
False muscular projection, 659
passage by probe, 227, 236
Family ataxia, 742
Far point, 68
Fascia, oculo-orbital, 11
inflammation of, 632
tarso-orbital, 196, 197, 205, 211
Fascicular keratitis, 273
palsies, 666
Fat and fatty degeneration, demonstration of, 786
Fatigue-field, 746
FEHR, 243
Feigned blindness, 747
FERRI, 643
FERRY, 219
Fetal eye-cup, 4
lens, dimensions of, 51
optic nerve, 5
retina, 5
vitreous humor, 3
Fibres of crystalline lens, 52
of Müller, 44
of optic commissure, 47
Fibrillary contraction of orbicularis muscle, 189
Fibrin, demonstration of, 787
Fibrinous iritis, 385
Fibroma of conjunctiva, 240
of cornea, 305
of eyelids, 175
Fibromatosis nervi optici, 530
Fibrous coat of eye, 29
FICK, 345, 385
Field of fixation, 658
of vision, 77
examination of, 124
in glaucoma, 569, 570, 572, 576
in optic-nerve atrophy, 545
in optic neuritis, 533
in toxic amblyopia, 542
method of examining, 124
reversal of, 558
Fifth nerve, 18
Filamentary keratitis, 332
Filaria in anterior chamber, 396
in lens, 471
Filaria loa, 285
subconjunctival, 285
FILEHNE, 745
Filix mas, amblyopia from, 542
Filtration angle. See *Iridocorneal angle.*
chemosis, 293
edema, 457
FINSEN'S light, 177
First principal focus, 63
FISCH, C., 181
FISCHER, 496
FISCHER, W. E., chapter contributed by, 772
FISHER, W. A., 371, 372
Fissure, fronto-maxillary, 159
sphenoidal, 9
spheno-maxillary, 10
Fistula, lacrimal, 227
of eyelid, 159
of lacrimal gland, 221
of the cornea, 342, 346
spurious, 346
treatment of, 347
true, 346
Fistulous staphyloma, 346
FITZGERALD, 642
Fixed cells of cornea, 30
Fixing eye specimens, 773
FLEMMING'S solution, 775
FLEXNER, 482
Flimmerskotom, 557
Fluorescin, 93, 292
Focal lesions of the brain, 737
Focus, anterior principal, 63
first principal, 63
posterior principal, 62
second principal, 63
Follicles in drug conjunctivitis, 259
Follicular conjunctivitis, 254, 257
diagnosis, 258
etiology, 258
pathology, 258
prognosis, 258
symptoms, 258
treatment, 258
ophthalmia, 257
trachoma, 257
Folliculosis, 257
FONTANA, spaces of, 31, 41
Foramina of orbit, 9
Foreign bodies in anterior chamber, 396
in ciliary body, 409
in conjunctiva, 294
in cornea, 354
in crystalline lens, 472
in eyeball, 370
in eyelids, 195
in iris, 395
in lacrimal passages, 223
in orbit, 650
in sclera, 370
in vitreous humor, 480
localization of, 153
magnetic, 371
non-magnetic, 373
Formalin, 266
Formol, 774
-Müller fluid, 774
Form-sense, 118
Fornix, examination of lower, 89
examination of upper, 89
FÖRSTER, 423, 467, 501, 509, 510
Fourth nerve, 18
paralysis of, 734
Fovea centralis, anatomy of, 42, 46
reflex of, Plate V, 61
FOX, L. W., 460, 615, 685
Fracture of ethmoid bone, 293, 649
of lamina cribrosa, 649
of optic canal, 649
of orbital bones, 648
FRANCISCO, 268
FREUND, 739
FRICKE'S blepharoplasty, 213, 214
FRIDENBERG, 152, 513
FRIEDENWALD, 387, 558
FRIEDREICH'S ataxia, 742
FRIES, 553, 555
FROMAGET, 221
Frontal bone, 8
nerve, 18

Frontal sinus, diseases of, 633
Fronto-maxillary fissure, 159
FROST, 421, 499, 515, 612
  method of implantation of sphere, 612
Frühjahr's catarrh. See *Vernal conjunctivitis.*
FUCHS, 41, 164, 200, 286, 287, 288, 360, 415, 418, 486, 492, 498, 513
Fuchsin, 781
Fugacious episcleritis, 363
Functions of the iris, 70
  of the retina, 77
Fundus oculi, anatomy of, 56
  appearance of normal—Plates II, III, and IV
  examination of, 140
  normal, 56
  of albino—Fig. 2, Plate III
  of brunette—Fig. 2, Plate II
  variations in, 59
Fundus-reflex test, 144
Fungus hæmatodes oculi, 482, 484
  trichophyton, 186
Fusion of images, 674

GAILLARD, 206
GALEZOWSKI, 220, 250, 299, 389, 496, 498, 505, 522
GALLENGA, 262
Gamma, angle, 69, 713
GAMO PINTO, 316
Ganglion, ciliary, 17
  Gasserian, 164, 200, 317
  inferior cervical, 19
  lenticular, 17
  middle sympathetic, 19
  of Meckel, 19
  ophthalmic, 17
  superior cervical, 19
Gangrene of eyelids, 179
GARGARIN, 315
GASPARINI, 252
GAY, 550
GAYET'S iridectomy, 399
  operation, 355, 358
Gelsemium, 190
General paralysis of the insane, 743
GENSOUL, 229
GÉRARD-MARCHANT, 584
GERLACH, illustration from, 12
Gerontoxon corneæ, 349
GESCHEIDT, 489
GESSNER, 641
Giant magnet, 371
GIFFORD, 251, 252, 256, 275, 276, 498, 605, 615
  reflex, 98
GILFILLAN, 418
GILLET DE GRANDMONT, 199, 525
GIMURTO, 517
Glands, acino-tubular, 26
  cervical, 289
  ciliary, 38
  lacrimal, 7, 22
  of Krause. See *Glands, acino-tubular.*
  of Meibomius, 26
  of Moll, 26
  of Waldeyer, 26
  of Zeiss, 166
  pre-auricular, 173, 176, 184, 263, 275, 289, 290
  submaxillary, 176, 184, 275, 289
Glass eyes. See *Artificial eyes.*
  shield after symblepharon operation, 299
  vitreous, 611
Glasses. See *Lenses* and *Spectacles.*
Glaucoma, 418, 562
  absolutum, 580
  acute inflammatory, 577
  after cataract operation, 458
  anatomic changes in, 565
  anterior, 569
  anterior chamber in, 568
    ciliary arteries in, 567
    ciliary veins in, 567
  anterior sclerotomy in, 591
  aqueous humor in, 569
  arecolin in, 576
  atropin in, 585
  cataract in, 568, 569
  cervical sympathetic in, 565
  changes in intra-ocular contents in, 569
  choroid in, 568
  chronic irritative, 576
  ciliary body in, 568
Glaucoma, clinical varieties of, 572
  cocain in, 576
  congenital, 582
  cornea in, 567
  crystalline lens in, 568
  cyclicotomy for relief of, 590
  de Wecker's method in, 592
  definition of, 562, 563
  diagnosis of, 573
  dilation of pupils in, 568
  enucleation for, 581
  eserin in, 576
  etiology of, 563, 578
  excision of cervical sympathetic ganglion in, 586
  eyelids in, 567
  general considerations of, 562
  halo in, 567
  Hancock's operation in, 590
  hemorrhages in, 577
  hemorrhagic, 493, 577
  heredity of, 563
  infantile, 582
  intra-ocular tension in, 562, 567, 572, 577, 580, 582, 584
  iridectomy in, 574, 577, 585
  iris in, 568
  lens in, 568
  macroscopic changes in eyeball in, 566
  massage in, 576
  miotics in, 576
  nasal disease in, 564
  operations for relief of, 584
  ophthalmoscopic signs of, 570
  optic nerve in, 569
  pain in, 572, 576, 577
  pathology of, 565, 578
  pilocarpin in, 576
  posterior, 569
  posterior sclerotomy in, 591
  post-iritic, 387
  prevention of, 581
  prognosis of, 579
  Quaglino's method in, 591
  retina in, 569
  sclera in, 567
  sclerotomy in, 591
  secondary, 458, 584
  signs of, ophthalmoscopic, 570
  simplex, 572
  sympatheticectomy for, 575, 577
  symptoms of, 572, 576 578
  treatment of, 574, 577, 580, 581
    unaffected eye in, 581
Glaucomatous cataract, 568
  degeneration, 580
  excavation, 570
  eyes, anatomic changes in, 567
Glioma of the retina, 482
Globe. See *Eyeball.*
GLORIEUX, 259
Glycogen, demonstration of, 790
Glycosuric cataract, 449
  retinitis, 503
GOLDBERG, HAROLD G., chapter contributed by, 756
GOLDZIEHER, 23, 275, 433
GOLGI, 43
GONIN, 549
Gonococcus, 267
Gonorrheal conjunctivitis, 262
  iridochorioiditis, 427
  ophthalmia of the adult, 263
    of the newborn, 267
GONZALES, 539
GORDON, 645
GOSSETTI, 270
GOULD, 377, 548, 551
Gouty retinitis, 511
GOWERS, 642, 734
GRADLE, 273, 274
Graduated tenotomy, 683
Grafting of skin, Italian method, 215
  le Fort's method, 216
  Ollier's method, 213
  Reverdin's method, 213, 217
  Tagliacozzi's method, 213
  Thiersch's method, 217
Grafts in ectropion operations, 208
  in pterygium operations, 297

Grafts in symblepharon operations, 299
GRAM-WEIGERT method, 791
GRANDCLEMENT, 268, 390, 393
Granular conjunctivitis, 277
Granulation, false, 258
  true, 258, 277
  tumors of conjunctiva, 241
Granuloma of conjunctiva, 241
  of iris, 380
GRATIOLET, radiations of, 82, 736
Grattage, 283, 301
GRAVES'S disease, 745
GREEFF, 45, 366
Green vision, 559
GRENACHER, 780
GRIFFITH, A. HILL, 339, 415, 426, 427, 642
GRIFFITH'S method, 776
Grill-like keratitis, 339
GRINDON, J., illustration from, 173
GRIZON, 525
GROENOUW, 263, 269, 504, 505, 510, 553, 554
GROSZ, 553
GRUENING, 531
GUIATA, 237, 420
Gumma of chorioid, 419
  of ciliary body, 405
  of conjunctiva, 292
  of eyelid, 173
  of iris, 386
  of orbit, 629
Gummatous iritis, 386
GUMPPER, 601
GUNN, 515, 540
GUNN'S dots, 515
GURWITSCH, 21, 508
Gymnastic exercise by prisms, 680

HAAB, 339, 372, 373, 380, 432, 494, 600
  magnet operations, 372
HACK, 642
HAGLUND, 269
Hair of caterpillar in conjunctiva, 285
Hair-optometer, 122
  use of, 122, 123, 124
HÅLA, 181
HALE, 390
Half-sight, 558
HALL, ERNEST, 611, 613
Halo in glaucoma, 567
HAMILTON, 471
HANCOCK'S operation for glaucoma, 590
HANN, 525
HANSELL, 252
Hardening of eye specimens, 777
HARLAN, 205, 298, 400
HARLAN'S operation for entropion, 205
  for symblepharon, 299
HARTLEY, 637
HARTRIDGE, 438
HASNER, 361
HASNER D'ARTHA'S blepharoplasty, 214
HAUG, method of, 776
HEAD, 164
Healing of corneal wounds, 353
HEBRA, 187
  ointment of, 187
HEINE, 67
HEISRATH'S operation, 299
HELMHOLTZ, 51, 66, 67, 82, 551
  ophthalmoscope of, 134
  theory of accommodation of, 66
HELMHOLTZ-YOUNG theory of color-perception, 551
Hemalaun, 779
Hemeralopia, 557
Hemianopsia, 736
  absolute, 736
  altitudinal, 736
  binasal, 736, 738
  bitemporal, 736, 738
  complete, 736
  homonymous, 736
    diagram illustrating, 737
  in focal brain disease, 738
  incomplete, 736
  inferior, 737
  lesions producing, 736
  superior, 737
Hemianoptic pupil-symptom of Wernicke, 739
  pupillary inaction, 97
Hemorrhage, amblyopia from, 553
  beneath conjunctiva, 293
  chorioidal, 433
  during cataract extraction, 454
  following cataract extraction, 455
  in glaucoma, 577
  into chorioid, 433
  into cornea, 353
  into orbit, 637, 646
  into retina, 492
  into sheath of optic nerve, 549
  into vitreous humor, 476
Hemorrhages in myopia, 712
Hemorrhagic glaucoma, 493, 577
  retinitis, 492
Hemostasis, artificial, 755
HENLE, 46, 55, 57
HENSCHEN, 736
HERBERT, 276, 281, 456, 457
HERING, 152
  experiment of, 152
  theory of color-blindness, 551
Hernia of Descemet's membrane, 308
  of iris, 395
  of orbital fatty tissue, 168
Herpes conjunctivæ, 274
  corneæ, 333, 336
  frontalis, 163
  zoster ophthalmicus, 163, 743
HERTEL, 490
HESS, 199
Heterochromia, 376
Heterograft, 216
Heterophoria, 670, 675
  diagnosis of, 675
  graduated tenotomy for, 683
  symptoms of, 679
  tests for, 675, 676, 677
  treatment of, 679
  varieties of, 679
Heterophthalmos. See *Heterochromia*.
Heterotropia, 670
Hetol, 302
HEUSE, 605
HEUSTIS, 242
HEYMANN, 349, 505
Hexagonal pigment-cells of the retina, 44
HIGGINS, 224, 642
HILBERT, 284
Hippus, 96, 396
HIRSCH, 512, 513
HIRSCHBERG, 417, 430, 479, 485, 503, 553, 555, 600
  magnet of, 371
HIRST, 617
HIS, 8, 65
History of cases, 85
  lives, 160
  HOFFMANN, 271
HOLDEN, 417, 513, 518, 519, 555
"Holes" in the macula, 522
HOLMES, C. R., 231, 284
Holocain, 255, 295, 754
Homatropin, 133
Hordeolum, 166
HORNER, 275, 345, 380, 546
  muscle of, 24
Horny growths. See *Cornu cutaneum*.
Horopter, 75, 76
HORTSMANN 522
Hot applications in conjunctivitis, 251, 254, 257, 270
  in dacryocystitis, 226
  in episcleritis, 363
  in iritis, 392
  in keratitis, 314
  in orbital cellulitis, 632
  in orbital periostitis, 629
  in scleritis, 366
"Hot eye," 363
HOTZ, 204, 205, 210, 211, 212, 213, 297, 299, 300, 494
  operation of, for ectropion, 210
    entropion, 204
HOWE, LUCIEN, 17, 510
HUIZINGA, 612
HULEN 10
Humor aqueous, 396
  vitreous, 54
    diseases of, 473

HUTCHINSON, 163, 388, 424
teeth, 323
Hyalin and colloid degeneration, demonstration of, 788
Hyalitis, 387, 473, 487
acute suppurative, 487
suppurative, 474
Hyaloid artery, 473
persistent, 473
membrane, 55
Hydrocephalus, 743
Hydrogen peroxid, 776
Hydrophthalmos, acquired, 582
congenital, 582
treatment of, 583
Hygiene of the eyes, 756
Hyoscin, 133
Hyoscyamin, 133
Hyperboloid lenses, 345
Hyperemia, conjunctiva, 248
of choroid, 699
of conjunctiva, active, 249
passive, 250
of optic-nerve head, 534
posterior conjunctival, 248
of retina, 489, 490
Hyperesophoria, 101, 670
Hyperesthesia of retina, 526
Hyperexophoria, 101, 670
Hyperidrosis, 179
Hyperkeratosis, 167
Hypermetropia, 692
absolute, 697
angle alpha in, 698
definition of, 692
diagnosis of, 699
diminishing, 692
etiology of, 694
facultative, 697
in strabismus, 698
latent, 697
manifest, 697
ophthalmoscopy in, 142
pathology of, 694
relative, 697
retinal image in, 696
symptoms of, 695
total, 697
treatment of, 700
Hypermetropic disc, 542
Hyperopia. See *Hypermetropia*.
Hyperostosis, 630
Hyperphoria, 670
Hypertrophy of lacrimal gland, 220
Hypertropia, 670
Hyphemia, 394
Hypometropia. See *Myopia*.
Hypopyon in corneal ulcer, 306
-keratitis, 311
source of, 306
Hypotony, 584
Hysteria, 746
Hysteric alopecia, 182
amblyopia, 526, 558, 746
blepharospasm, 189
blindness during the puerperium, 509

Ichthyol, 283
Ichthyosis, 280
Identical points, 75
Idiocy, amaurotic family, 512
Illumination by retinoscopy mirror, 93
focal, 93
oblique, 93
Image, inversion of, 81
retinal, 65
size of ophthalmoscopic, 139
Images, mechanism of formation of, 62
produced by lenses, 689
Imbedding, 777
Impetigo, 161
Implantation of artificial vitreous in sclera, 611
into Tenon's capsule, 612
cyst of iris, 377
Inch system of numbering glasses, 691
Index of refraction, 687
of air, 64
of aqueous humor, 64
of cornea, 64
Index of crystalline lens, 64
of reduced eye, 65
of vitreous humor, 64
of water, 64
Indications for enucleation, 606
Indirect ophthalmoscopy, 136, 137, 138
Indirect vision, 79
Inequality of pupils, 96
Infantile amaurotic family idiocy, 512
Infantile paralysis, 743
Infected wounds of eyeball, 370
Inferior oblique muscle, 15
paralysis of, 662
rectus muscle, 15
paralysis of, 662
Infiltration anesthesia, 754
Infra-orbital foramen, 10
nerve, 10
vessels, 10
Injected blood-vessels of iris, etc. (Fig. 35), 36
Injections, subconjunctival, 301
Injuries of choroid, 433
of ciliary body, 409
of conjunctiva, 294
of cornea, 351
of crystalline lens, 471
of eyelids, 193
of iris, 394
of optic nerve, 560
of orbit, 646
of retina, 521
of sclera, 368
Insanity, 743
Instruments, preparation of, 752
Insufficiency of ocular muscles. See *Heterophoria*.
Intermarginal line, 26, 202.
Internal rectus muscle, 14
advancement of, 684
paralysis of, 661
tenotomy of, 681
Interorbital space, 11
Intracranial tumor, 508, 534
Intra-ocular cysticercus, 381, 418 478, 487
hemorrhage, 394, 433, 454, 455, 476, 492, 494, 521
injections, 605
tension, normal, 562
tumors, 377, 378, 380, 404, 414, 482
Inversion of the image, 81
of the iris, 395
Inverted image of fundus, 136
Iodic acid, 283
Iodid of potassium, 292, 293, 497, 511, 666
Iodin injections in retinal detachment, 525
Iodoform, 290, 301, 475
amblyopia from, 542
in lymphoma of the conjunctiva, 276
use of, in anterior chamber, 433, 456
IONA, 270
Iridal part of the retina, 46
Iridectomy, 397, 585
accidents during, 399
Deschamps's method, 400
Gayet's method, 399
in aphakic eyes, 400, 585
in cataract extraction, 453, 458
in corneal diseases, 316, 330, 336, 343, 348, 351, 353
in corneal opacity, 351
in detachment of the retina, 525
in glaucoma, 399, 585
in hemorrhagic glaucoma, 580
in hydrophthalmos, 583
in iritis, 393
in juvenile cataract, 463
in obliteration of the anterior chamber, 399
in prolapse of iris, 395
in pterygium, 287
in sympathetic ophthalmitis, 605
in uvéite irienne, 393
instruments for, 398
intra-ocular hemorrhage after, 586
method of performing, 398
optical, 397
in cataract extraction, 458
preliminary to cataract extraction, 399, 459
therapeutic, 398
Iridenkleisis, 394, 400
Irideremia. See *Aniridia*.
traumatic, 394, 395

Iridesis. See *Iridodesis.*
Iridocapsulotomy, 401
Iridochorioiditis, 387, 427
  gonorrheal, 427
Iridocorneal angle (Fig. 31), 32
Iridocyclitis, 387, 418, 595
  after cataract extraction, 456
  endogenous, 409
  plastica, 408
  serous, 406
  spontaneous chronic, 606
  sympathetic. See *Sympathetic ophthalmitis.*
Iridocystectomy, 402, 403
Iridodesis, 397
Iridodialysis, 394, 400, 405
Iridodonesis, 396
Irido-ectomy, 402
Iridoplegia. See *Paralytic mydriasis.*
Iridotomy, 400
Iris, absence of, 376
  absence of pigment in, 376
  adhesion of, to the cornea, 377
  anatomy of, 33
  anomalies of, 374
  anteversion of, 394
  arteries of, 34
  blood-vessels of, 34, 35
  bombé, 387, 400
  bridge coloboma of, 374
  cholesteatoma of, 378
  chromatic asymmetry of, 376
  coloboma of, 374
  color of, 33
  complete detachment of, 394
  congenital anomalies of, 374
  congenital atrophy of, 377
  congenital tumors of, 377
  contraction of, 71, 96
  cysticercus of, 381
  cysts of, 377
  development of, 4
  -diaphragm, 147
  dilation of, 71, 96
  dilator muscle of, 34
  diseases of, 374-403
  displacement of. See *Injuries of the iris.*
  exfoliation of, 376
  filaria of, 381
  foreign bodies in, 395
  functional disorders of, 396
  functions of, 70
  granuloma of, 380
  gummatous inflammation of, 386
  hyperemia of, 382
  implantation cysts of, 377
  -inaction of Wernicke, 97, 739
  incarceration of. See *Prolapse of iris.*
  inflammation of, 381
  injuries of, 394
  inversion of, 395
  laceration of sphincter of, 394
  leprosy of, 380
  lymphomata of, 380
  melanoma of, 376
  mobility of, 96
  motor disturbances of, 396
  muscle-fibres of, 33, 34
  myomata of, 380
  myosarcomata of, 380
  nerves of, 35
  nevus of, 380
  operations on, 397
  papilloma of, 376
  paralysis of, 96, 668
  pearl-cysts of, 378
  physiology of, 70-72
  pigment of, 34
  prolapse of, 395
    after cataract extraction, 454
  radial section of (Fig. 33), 34
  retention cysts of, 377
  rupture of, 394
  sarcoma of, 378
  serous cysts of, 377
  sphincter of, 34
  total prolapse of, 309
  transfixion of, 400
  tremulous. See *Iridodonesis.*
  tubercle of, 380
  tumors of, 377
Iris, unusual tumors of, 380
  vascular tumors of, 380
  wounds of, 394
Iritis, 265, 289, 291, 293, 381
  after cataract operations, 456
  causes of, 388
  complications of, 386, 387
  congenital, 388
  diabetic, 389
  diagnosis of, 383
  etiology of, 388
  fibrinous, 385
  gonorrheal. See *Gonorrheal iridochoriotiditis.*
  gummatous, 386
  infectious, 393
  iridectomy in, 393
  operative treatment of, 393
  papulosa, 386
  parenchymatous, 385
  pathology of, 386
  plastic, 384
  prognosis of, 390
  purulent, 385
  rheumatic, 388
  senile, 389
  serous, 384
  sequelæ of, 387
  spongy, 385
  suppurative, 385
  sympathetic, 599
    serous, 599
  symptoms of, 381
  syphilitic, 386
  traumatic, 388
  treatment of, 391
  tubercular, 386
  varieties of, 384
Iritomy, 400
Iron, demonstration of, 790
Irradiation, 81
Irregular astigmatism, 723
Irritation of the retina, 526
Ischemia of retina, 489
Isolated dots in the retina, 515
IWANOFF, 281
  illustrations from, 39

Jaborin, 576
JACKSON, E., 94, 135, 747, 753
  binocular magnifier of, 94, 95
JACKSON, HUGHLINGS, 537
JACOB'S ulcer, 176, 177
JACOBSON, diffuse syphilitic retinitis of, 501
JAEGER, 58, 203, 425
JAENCKE, 525
JAESCHE, 300
JAESCHE-ARLT operation for trichiasis, 202
Jamaica ginger, amblyopia from, 544
JANSSON, 157
JAVAL, 73
JAVAL-SCHIOETZ ophthalmometer, 116
JENNINGS, J. E., illustrations from, 78, 82, 571
JENSEN, 525
Jequirity, 269, 284
JESSUP, 496
JEUNET, 584
JOCQS, 467, 468
JOHNSON, 371, 583
JOHNSON, G. LINDSAY, 60, 412
JONNESCO, 584
JUDA, 243
JULER, 258
Junction, corneoscleral, 40
JUNG, 486
JUNIUS, 252
Juvenile cataract, 463

KAGENAAR, 177
KAISERLING'S method, 773
KALT, 225, 459
  suture, 459
Kataphoria. See *Cataphoria.*
Katatropia. See *Catatropia.*
KEBER, illustration from, 174
Keloid, 174
Keratectasia, 346
Keratite en bandelette, 334
  quadrillée, 339
Keratitis, 305

Lead acetate, 255
encephalopathy, 508
LEBER, 35, 243, 284, 389, 419, 507, 508, 519, 523, 555, 562, 603
LEDERER, 641
Leeches in iritis, 392
LEHMANN, 509
LENHOSSEK, 4
Lens, 688
biconvex, 63
concave, 688
conjugate focus of a convex, 689
convex, 63
conjugate foci of, 689
crystalline. See *Crystaline lens.*
cylindric, 690
focal distance of a, 688
focus of a concave, 690
formation of images by a, 689
optical centre of a, 688
position of image formed by a convex, 690
principal focus of a concave, 690
of a convex, 688
toric, 680
Lenses, combination of, 691
cylindric, 690
numbering of, 690
properties of optical, 686
spheric, 688
toric, 690
Lenticonus, 438
anterior, 438
posterior, 438
Lenticular ganglion, 17
Lentiglobus, 438
LEONARD'S method of x-ray localization, 151
LEOPOLD, I., illustrations from—Fig. 1, Plate XIX, and Fig. 1, Plate XX.
Leprosy of conjunctiva, 290
of cornea, 341
of eyelids, 174
of iris, 380
Leptomeningitis, 740
Leptothrix in canaliculi, 223
LERCHE, 484
Leucocythemic retinitis, 503
Leucoma, adherent, 350
Leucosarcoma of chorioid, 486
Leucosis, 412
Levator palpebræ superioris muscle, 13
contraction of, with pterygoid, 191
LÉVEILLÉ, 16, 17
LEWIN, 642
LIEBREICH, 421, 505
Ligamentum pectinatum, 31
Light, definition of, 686
perception of, 121
-reflex of retinal blood-vessels, 58
refraction of, 687
-streak, 58
waves of, 686
Light-perception, qualitative, 121
quantitative, 121
Light-sense, testing of, 127
in optic-nerve atrophy, 546
subjective sensations of. See *Photopsia.*
LILIENFELD, 576
Limbus of cornea, 30
Line of fixation, 68
Linear extraction of cataract, 460
method of measuring squint, 673
Lipoma of eyelids, 168
of orbit, 621
subconjunctival, 240
Lipomata cavernosa, 621
telangiectodes, 621
LIPPINCOTT, 371
Lippitudo, 186
LISTING, law of, 73
reduced eye of, 65
Lithia-carmin, 781
Lithiasis of conjunctiva, 276
Liver, use of, in nyctalopia, 557
LLORET, 300
Localization of foreign bodies in the eye, 153
Locomotor ataxia, 741
Long ciliary arteries, 20
LORING, E. G., 526
ophthalmoscope of, 135
LORING, F. B., 297
Lupus of conjunctiva, 291
of eyelids, 173
vulgaris of eyelids, 173
Luxation of crystalline lens, 439, 469
of eyeball, 647
Lymphadenoma of lacrimal gland, 221
Lymphangioma of caruncle, 296
of eyelids, 175
of orbit, 623
Lymphatics of eyelids, 28
Lymph-channels of the cornea, 30
of the eye, 55
perivascular, 60
-space, intervaginal, of optic nerve, 56
perichorioidal, 56
Lymphectasia of conjunctiva, 243
menorrhagica, 243
Lymphoma of conjunctiva, 275
of eyelids, 175
of iris, 380

MACEWEN, 636, 637
MACKENZIE, 594
MACKENZIE, S., 489
MACNAMARA, 519
Macula corneæ, 350
lutea, Plate V, 42, 43, 46, 56, 60, 79
halo around, 60
"holes" in, 60, 522
physiology of, 79
symmetrical changes in, 512
traumatic perforation of, 522
vascularity of, 60
Macular colobomata, 412
keratitis, 333
region, colloid disease of, 435
Madarosis, 186
MADDOX, double prism of, 104
rod test, 675
MAGENDIE, 316
MAGNANI, 262
Magnet, giant, 371
Magnification of ophthalmoscopic image, 139
MAGNUS, 492
Malar bone, 8
Malarial keratitis, 331
MALHERBE, 361
Malignant pustule of eyelid, 162
Malingering, 556
tests for, 151
MANDELSTAMM, 555
Manifest hypermetropia, 697
MANZ, 5, 158, 537
MANZ-ZIEGLER, illustration from, 5
MARCHI, method of, 784
Marginal phlyctenular keratitis, 320
ulcerative keratitis, 337
MARPLE, 496, 498
MARTIN, W. A., 337
Massage in retinal embolism, 497
of cornea, 350
of optic nerve, 548
MAUNTHER, 60, 552
MAY, 135, 299
MAYOU, 284
McQUEEN, 269
McREYNOLDS, 297
Measurement of astigmatism, 117, 142, 149
Measurements of the crystalline lens, 51
of the eyeball, 29, 692
Mechanical exercise in ocular paralyses, 668
injuries of the cornea, 352
Media, location of opacities in the transparent, 139
Medullated fibres of optic nerve, 48
"Medusa head" nerve, 534
Megalocornea, 304, 345
Megalophthalmos, 304
Megalopsia, 500, 559
Meibomian cysts. See *Chalazion.*
glands, 26, 276
MEIBOMIUS, glands of, 26
MEIGS, A. V., 415
Melanin, 416
Melanoma of iris, 376
Melanosarcoma of chorioid, 419
Melanosis of sclera, 361
MELLINGER, 315
Membrana limitans externa, 43
interna, 43

Membrana vasculosa, 43
Membrane of Demours, 31
of Descemet, 31, 246
MÉNARD, 159
Meningitis, cerebrospinal, 740
following enucleation, 610
tubercular, 740
Meningocele, 618
Meniscus, 688
Mental blindness. See *Mind-blindness* and *Psychic blindness.*
Mercury in iritis, 392
in optic-nerve atrophy, 547
MERKEL, 11, 13, 49, 50, 55
MERKEL and KALLIUS, 20
Metallic dots in the retina, 515
foreign bodies in the eyeball, 370
Metamorphopsia, 421, 559
varians, 559
Metastatic carcinoma of chorioid, 414
sarcoma of chorioid, 415
Method of inverted image. See *Indirect ophthalmoscopy.*
of upright image. See *Direct ophthalmoscopy.*
Methyl alcohol, amblyopia from, 544
Methylene blue, 791
Metre angle, 123
Metric system of numbering lenses, 690
MEYER, 427, 603
MEYER, P., 779
MEYNERT'S fibres, 47, 739
MIAL, 231
Microblepharon, 159
Microphakia, 438
Microphthalmos, 411
Micropsia, 500, 501, 559
Microsporon trachomatosum, 277
Middle cervical ganglion, 19
Migraine, 557, 743
ophthalmic, of Charcot, 791
Miliaria, 180
Miliary aneurisms of retina, 429
tubercle of chorioid, 412
Military conjunctivitis, 276
ophthalmia, 277
Millium, 180
MILLES, 480
MILLIKIN, 418
Mind-blindness, 739
Miners' nystagmus, 669
Miosis, 96, 396, 734
irritative, 96, 734
paralytic, 96, 669, 734
Miotics, 134
Mirror, concave retinoscopy, 137
ophthalmoscopic, 134, 136
plane retinoscopy, 150
MITTENDORF, 467, 473
MITVALSKI, 414
Mobility of the eye, 72
of the iris, 96
MÖBIUS, 643
MOLL, glands of, 26
Molluscum contagiosum, 172
epitheliale, 172
Monocular diplopia, 72
Monoculus, 754
MONTGOMERY, 166
MONZON, 378
MOORE, W. O., 503
MOOREN, 467, 515, 597, 598, 644
ulcer of, 310
MORAX, 251, 252, 256
MORGAGNI, spheres of, 442
Morgagnian cataract, 442
Morphin, 255
MORTON, 612
MORVAN'S disease, 744
MOTAIS, 199
Motor disturbances of iris, 396
Motor oculi nerve, 17
Mounting of sections, 784
Mucocele, 226
of frontal sinus, 634
of inferior canaliculus, 223
Mucoid degeneration, demonstration of, 788
MULES, 198, 596, 611
method of evisceration, 611
advantages of, 613
Mules's operation for ptosis, 198
MÜLLER, fibres of, 44
muscle of, 12
MÜLLER, L., operation for ectropion, 210
MULLER'S fluid, 774, 775
Multiple neuritis, 742
sclerosis, 740
Muscæ volitantes, 70, 475, 476
Muscle, ciliary, 36, 37
oblique inferior, 15
superior, 13
of Horner, 24
of Müller, 12
of Riolan, 26
rectus externus, 14
inferior, 15
internus, 14
superior, 13
Muscles, action of the, 654
Muscular asthenopia, 679
insufficiency. See *Heterophoria.*
tissue of the iris, 33
MUTTERMILCH, 277
Mycotic keratitis, 331
Mydriasis, 396, 734
paralytic, 668, 735
Mydriatics, 131
in iritis, 382, 391
Myelitis, 732
Myoma of chorioid, 420
Myopia, 703
acquired, 709
angle gamma in, 713
atrophic changes in, 425
axial, 367, 704
chorioidal changes in, 425, 712, 714
congenital, 706
conus in, 710
correction of, 716
curvature, 705
definition of, 703
detachment of retina in, 710
diagnosis of, 714
etiology, 704
heredity in, 706
in school-children, 707
influence of muscles in producing, 706
ophthalmoscopic appearance in, 714
ordering glasses for, 715, 716
pathologic evolution of, 768
pathology of, 710
prevalence of, in public schools, 767
prisms in, 716
reading glasses in, 716
removal of crystalline lens in, 716
retinal hemorrhages in, 714
skiascopy in, 148
symptoms of, 712
tenotomy of external rectus in, 716
theories of causation of, 705
transient, in iritis, 393
treatment of, 714
Weiss's reflex in, Fig. 2, Plate XX, 714
with glaucoma, 571
Myopic astigmatism, 719
chorioiditis, 425
crescent. See *Conus.*
degeneration, 426
Myosis. See *Miosis.*
Myxoma of conjunctiva, 241
of cornea, 304
of lacrimal gland, 221

Nævus pigmentosus, 170
vasculosis of eyelid, 175
NAGEL, 502
Nasal artery, 21
duct, anatomy of, 10, 24
diameter of, 24
obstruction of, 227
stricture of, 227
Naso-lacrimal canal, operations on the, 233
NATANSON, 262
Near point, 68
determination of, 122
of convergence, 123
-sight. See *Myopia.*
Nebula, 350
Necrosis of orbit, 627
Needle operation for cataract. See *Discission.*

Negative convergence, 123
NEISSER, 262, 264
Nephritis, retinitis in, 505
Nerve, optic. See *Optic nerve.*
  ophthalmic. See *Ophthalmic nerve.*
Nerves, corneal, 31
  iridal, 35
  palpebral, 28
Nervous diseases, ocular manifestations of, 733
NESNAMOFF, 283
NETTLESHIP, E., 427, 435, 504, 516, 773
Neuralgia, 743
Neurasthenia, 745
Neurasthenic asthenopia, 526
Neurectomy, opticociliary, 561
Neuritic dots in the retina, 515
Neuritis, descending, 536
  optic. See *Optic neuritis.*
  retrobulbar. See *Retrobulbar neuritis.*
Neuroepithelioma of the retina, 418
  diagnosis of, 484
  etiology of, 483
  pathology of, 482
  prognosis of, 487
  symptoms of, 484
  treatment of, 488
Neuroepithelium, 46
Neurofibroma of eyelid, 175
Neuroma of eyelid, plexiform, 175
Neurons of the retina, 43
Neuroparalytic keratitis, 316
Neuropathic keratitis, 316
Neuroretinitis, 387, 534, 535
Neurosis following traumatism, 745
Neurotomy, opticociliary, 561, 604
NEUSTATTER, 558
NEVE, 290, 341, 380
Nevus of iris, 380
NICATI, 407
NIEDEN, 317, 641
Night-blindness, 556
Nitrate of silver, 254, 257, 259, 266, 270
Nitrobenzol, amblyopia from, 542
Nitroglycerin in optic-nerve atrophy, 548
Nodal point, 62
Nodular keratitis, 333
Noma of the eyelids, 179
Non-gonorrheal purulent conjunctivitis, 268
Non-magnetic foreign bodies, 373
Non-suppurative keratitis, 320
NORDENSEN, 508, 523
Normal fundus oculi, Plate III
  intra-ocular tension, 562
NORRIS and OLIVER, 399
Notched teeth, 323
NOYES, H. D., 221, 224, 300, 361, 578, 585, 594
Nuclear palsy, acute, 665
    chronic, 666
  stains, 779
Nuclei of ocular nerves, 49
Nucleus of abducens nerve, 51
  of crystalline lens, 52
  of oculomotor nerve, 50
  of patheticus nerve, 51
NUEL, 326, 329, 334, 369, 562
  stitch for scleral rupture, 369
Numbering of lenses, 690
Nummular keratitis, 333
NUSSBAUM, 360
Nyctalopia, 556
Nystagmus, 669, 734
  acquired, 669
  congenital, 669
  miners', 669
  treatment of, 669

OATMAN, 414
Oblique illumination. See *Focal illumination.*
  muscles, anatomy of, 13, 15
    insufficiency of, 677
    paralysis of, 662
    tests for, 677
Occlusion of the pupil, 387
Ocular ataxia, 741
  lymph-spaces, 55
  manifestations of nervous diseases, 733
  movements and binocular vision, 72
  muscles, abnormal balance of. See *Heterophoria.*
Ocular muscles, absence of, 618, 733
    action of, 73
    advancement of, 684
    associated paralyses of, 664, 733
    balance of, 101
    congenital defects of, 618
    insufficiency of. See *Heterophoria.*
    operations upon the, 681
    paralysis of external. See *Ophthalmoplegia.*
    physiology of, 72
    spasm of, 734
    tests for balance of, 102
Oculomotor nerve, paralysis of, 663
  radiations of, 50
Oculo-orbital fascia, anatomy of, 11
  inflammation of, 632
OETTINGEN, 287
OGILVIE, 522
OLIVER, C. A., 242, 489, 578, 579, 580, 605, 612
Onyx, 307
Opacities in cornea, 350
  in crystalline lens. See *Cataract.*
  in vitreous humor, 475
Opaque nerve-fibres in the retina, 481; Fig. 2, Plate IV.
Operating mask, 749
Operation room, 755
Operations, for cicatricial orbit, 615
  for glaucoma, 584
  for muscular insufficiency, 681
  for post-operative cataract, 465
  for traumatic cataract, 464
  on the conjunctiva, 296
  on the cornea, 355
  on the crystalline lens, 449
  on the eyelids, 195
  on the iris, 397
  on the optic nerve, 560
  on the orbit, 651
  on the lacrimal apparatus, 231
  on the retina, 525
  on the sclera, 590, 591
  on the sympathetic nerve, 586
  preparation for, 749
Ophthalmia. See *Conjunctivitis.*
  gonorrheal, of adult, 263
  migratoria, 594
  neonatorum, 262
  nodosa, 285
  tarsi, 185
Ophthalmic artery, aneurism of, 639
  migraine, 191
  vein, anatomy of, 21, 22
    thrombosis of, 636
Ophthalmitis, sympathetic. See *Sympathetic inflammation.*
Ophthalmoblennorrhœa, 262, 267
Ophthalmomalacia, 436
Ophthalmometer, description of, 116
  Kagenaar's model of, 117
  of Helmholtz, 116
  of Javal-Schioetz, 118
  use of, 116
Ophthalmometry, 115
Ophthalmophantome, 749
Ophthalmoplegia, 665
  acute, 665
  chronic, 666
  external, 666
  internal, 666
  total, 666
Ophthalmoscope as a refractometer, 141
  description of, 134
  direct method of using, 138
  formation of image in direct method of using, 137
  in indirect method of using, 137
  indirect method of using the, 138
  magnifying power of the, 139
  methods of use, 135
  of Couper, 135
  of Harlan, 135
  of Helmholtz, 134
  of Jackson, 135
  of Knapp, 135
  of Knauer, 135
  of Landolt, 135
  of Loring, 135
  of May, 135

Ophthalmoscope of Morton, 135
of Pyle, 135
of Randall, 135
of Snell, 135
of Thorner, 134
use of the, 139
Ophthalmoscopy, 134
direct, 137
indirect, 137
Optic axis, 62
Optic canal, fracture of, 649
Optic chiasma, 47
Optic commissure, 47
Optic disc, 57
Optic nerve, absence of, 528
anatomy of, 41
anomalies of, 528
aplasia of, 528
apoplexy of, 549
atrophy of, 545
causes of, 546
congenital, 528
diagnosis of, 547
etiology of, 546
hereditary, 548
ophthalmoscopic changes in, 546
pathology of, 547
primary, 545
prognosis of, 547
retinal type of, 546
secondary, 545
symptoms of, 545
treatment of, 547
visual field in, 545
colloid bodies in head of the, 532
coloboma of sheath of, 528
congenital defects of, 528, 529
crossing of fibres of, 47
development of, 5
diseases of, 528-561
drusenbildungen in, 532
edema of head of, 536, 569
embolic atrophy of, 546
excavation of, in atrophy, 546
in glaucoma, 570
physiologic, 57
extradural tumors of, 531
hemorrhage into sheath of, 549
hereditary atrophy of, 548
hyalin bodies in head of, 532
hyperemia of, 534
injuries of, 560
intervaginal space of, 48
intracranial part of, 41
intradural tumors of, 530
lymph-channels of, 48
number of fibres in, 48
opaque fibres of, 481
operations on, 560
orbital portion of, 48
pallor of temporal segment of, 542
physiologic excavation of, 57
pigmentation of head of, 529
primary atrophy of, 545
radiations of, 49
resection of, 560
retinal type of atrophy, 546
secondary atrophy of, 545
sheaths of, anatomy of, 48
dilation of, 536
tumors of, 530
Optic neuritis, 533
after orbital affections, 631, 636, 638, 646, 649
albuminuric, 506
back-water theory of, 536
course and complications of, 536
descending, 534
diagnosis of, 535
etiology of, 535
forms of, 534
from brain tumor, 536
from general diseases, 535
hemorrhages in, 534
inflammatory theory of, 537
intra-ocular, 533
lymph-space theory of, 537
mechanism of, 536
monocular, 535
orbital, 539
Optic neuritis, Parinaud's theory of, 538
pathogenesis of, 536
pathology of, 536
prognosis of, 536
retrobular, 539
acute, 539
causes of, 539
chronic, 541
diagnosis of, 540
pathology of, 540
prognosis of, 540
symptoms of, 540
treatment of, 540
toxic, causes of, 542
ophthalmoscopic picture in, 542
second attacks of, 536
Sourdille's theory of, 538
spurious, 541
surgical treatment of, 539
sympathetic, 601
symptoms of, 533
syphilitic, 536
treatment of, 539
types of, 534
vasomotor theory of, 538
with dropping of fluid from nose, 541
Optic radiations, 49
Optic tract, 535
Optical axis, length of, 29, 692
Optical centre of lens, 688
Optical defects of normal eye, 68
Optical lenses, properties of, 686
Opticociliary neurectomy, 560
neurotomy, 560
Optograms, 80
Ora serrata, 42
Orange G, 782
Orbicularis, paralysis of. See *Lagophthalmos.*
pupillary reaction. See *Gifford's reflex.*
spasm of, 189, 734
Orbit, abscess of, 610, 627, 631
anatomy of, 8
aneurism by anastomosis in, 639
angioma of, 621
axis of, 8
blood-cyst of, 626
-vessels of, 20
bony tumors of, 624
capillary angiomata of, 621
carcinoma of, 625
caries of, 627
cavernous angiomata of, 621
cellulitis of, 630
cerebral symptoms following injury of, 649
chondroma of, 623
congenital anomalies of, 616
congenital cyst of, 618
contusion of, 646
cysts of, 618, 625
dermoid cyst of, 619
diseases of, 616-653
distension of cavities adjacent to, 633
echinococcic cyst of, 626
emphysema of, 648
enchondroma of, 623
endothelial tumors of, 625
enophthalmos, 640
epithelial tumors of, 625
examination of, 100
exenteration of, 653
exostoses of, 630
extirpation of whole contents of, 652
extravasation cyst of, 626
exudation cysts of, 625
fistula of, 627
follicular cysts of, 626
foreign bodies in, 650
fracture of, 648
of apex of, 649
gummata of, 629
hematema of, 626
hemorrhage into, 637
hydatid cyst of, 626
hyperostosis of, 630
inflammation of oculo-orbital fascia, 632
of sinuses adjacent to, 633
injuries of, 646
intermittent exophthalmos, 640
lipoma of, 621
lymphangioma of, 623

Orbit, necrosis of, 627
nevus of, 621
operations on, 651
osteoma of, 624
parasitic cysts of, 626
penetrating wounds of, 646
periostitis of, 627
periostosis of, 629
phlegmon of, 630
plexiform angiomata of, 621
neuroma of, 625
pulsating exophthalmos, 638
removal of tumors of, 651
by Krönlein's operation, 652
retention cysts of, 626
sarcoma of, 624
shape of, in myopia, 713
tumors of, 618
etiology of, 619
prognosis of, 620
symptoms of, 620
treatment of, 620
varieties of, 621
vascular tumors of, 621
wounds and injuries of, 646
Orbital cellulitis, 630
deep incisions in, 632
effects of, on vision, 632
following tenotomy, 682
prognosis of, 631
treatment of, 632
Organic entropion, 280
ORTH, 781
Orthophoria, 670
Orthoptic exercises in heterophoria, 680
training in strabismus, 674
Ossification of choroid, 420
Osteoma of conjunctiva, 242
of orbit, 624
OSTWALT, 432
O'SULLIVAN, 527
Ozena bacillus, 225

Pachymeningitis, 508
PAGENSTECHER, 199, 305, 460, 599
Pagenstecher's salve. See *Yellow oxid of mercury.*
operation for ptosis, 199
Palpebral ligaments, 26
PANAS, 197, 245, 305, 357, 471, 681
operation for ptosis, 199
Pannus, 325
crassus, 326
eczematosus, 320
granular, 328
jequirity in, 329
sarcomatosus, 326
tenuis, 326
treatment of, 328
Panophthalmitis, 264
ectogenous, 430
diagnosis of, 432
pathology of, 431
prognosis of, 432
symptoms of, 431
treatment of, 432
endogenous, 429
diagnosis of, 430
prognosis of, 430
symptoms of, 430
treatment of, 430
enucleation during, 433
evisceration during, 433
meningitis from, 433
Papilla. See *Optic nerve.*
Papillitis (see also *Optic neuritis*), 533
Papilloma of conjunctiva, 241
of cornea, 304
Papilloretinitis, sympathetic, 601
Papular conjunctivitis, 275
PAQUELIN cautery, 623
Paracentesis corneæ, 355
in corneal ulcer, 315
in iritis, 392
of cornea, 355
Paradoxic pupil, 743
Paraffin-imbedding, 777
injections, danger of, 207
Parallax test, 143
for heterophoria, 676
Parallax test in glaucoma, 571
Paralysis agitans, 743
bulbar, 645, 742
of accommodation, 669
of external rectus, 661
of inferior oblique, 662
of internal rectus, 661, 662
of ocular muscles, 657
cerebral, 664
clinical method of determination, 663
cortical, 664
diagnosis of, 661
diagrams illustrating, 658, 659, 660, 661, 665
diplopia in, 657
etiology of, 664
false muscular projection, 659
fascicular, 664
general symptoms of, 657
limitations of movements of globe, 658
nuclear, 664
orbital lesions in, 667
peripheral, 666
position of head in, 659
primary deviation in, 657
prognosis in, 668
projection of false image, 657
recurrent third-nerve, 667
secondary deviation in, 657
special symptomatology of, 661
treatment of, 668
of oculomotor nerve, 663
of superior oblique, 662
of superior rectus, 662
of third nerve, 667, 734
pseudobulbar, 742
Paralytic miosis, 669
mydriasis, 668
Parasitic skin diseases, 182
Parenchymatous iritis, 385
Parenchymatous retinitis, 502
Paresis of accommodation, 669
PARINAUD, 199, 251, 275, 538
conjunctivitis of, 275
PÄSSLER, 642
Patheticus nerve, 18
radiations of, 51
PAYNE, R. W., 437
Pearl-cysts of iris, 378
Pectinate ligament, 40
Pegged teeth, 323
Pemphigus, 165
of conjunctiva, 275
of cornea, 329
of eyelids, 165
Periectomy, 301
Perifolliculitis, 166
Perimeter, 124
Dana's, 125
McHardy's, 125
Skeel's, 125
Smith's, 125
Perimetry, 124, 125
Periostitis of orbit, 627
Periostosis of orbit, 629
Peripheral paralysis of ocular muscles, 666
Peritomy, 301
Perivascular lymph-channels, 60
PERL'S reaction, 790
Persistent pupillary membrane, 374
PETIT, 312
PFITZNER, 781
PFEIFFER, 277
PFLANGE, 519
PFLÜGER, 345
Phagedenic ulceration of eyelids, 179
Phlebosclerosis, 511
Phlegmon of lid, 162
Phloroglucin method, 776
Phlyctenula pallida, 260
Phlyctenular conjunctivitis, 272
keratitis, 320
ophthalmia, 272
pannus, 328
ulcer, 320
Phosphoridrosis, 179
Photophobia, 247
Photopsiæ, 421
Phtheiriasis, 182
Phthisis bulbi, 264, 436
Physiologic excavation, 57

Physiology of vision, 62
Plan, 161
Picric acid, 776, 782
Pigment patches in conjunctiva, 280
on optic disc, 529
streaks on the fundus, 518
Pigmentary degeneration of the retina, 519
Pigmentation of conjunctiva, 280
Pigmented retina and chorioiditis, 519
Pilocarpin, 576
in glaucoma, 576
Pince-ciseaux of de Wecker, 401, 402, 403
PINCUS, 304
Pinguecula, 287
"Pink eye," 252, 254
PLACIDO'S disc, 93, 344
Plastic chorioiditis, 420
cyclitis, 408
iritis, 384
Plexiform neuroma of eyelid, 175
Plica semilunaris, 25
affections of, 295
Pneumococcus, 252, 269
conjunctivitis, 252
in corneal ulcers, 309, 312
POLLOCK, illustration from, 403
Polycoria, 375
Polypi of conjunctiva, 241
PORTER, W. H., 505
POSEY, 489
POST, M. H., 418
Posterior polar cataract, 426
sclerochorioiditis, 425
sclerotomy, 593
synechiæ, 387
staphyloma, 425, 711
symblepharon, 280
Postero-lateral sclerosis, 742
Post-iritic glaucoma, 387
Post-operative cataract, 446
operations for, 465
Poultices, 251
POWERS, 462
PRAUN, 368
Pregnancy kidney, 510
PRENTICE'S method of numbering prisms, 688
Presbyopia, 725
definition of, 725
diagnosis of, 728
etiology of, 725
selection of glasses, 728
symptoms of, 726
treatment of, 728
Pretended amblyopia, 556
Primary deviation, 657
opacity of the cornea, 334
Principal anterior focus, 63
axis, 688
focal distance, 688
posterior focus, 62
Prism, angle of deviation of a, 687
convergence, 108
Crétès's, 107
dioptre, 688
divergence, 109
holders, 109
Maddox's double, 104
refracting angle of a, 688
Risley's, 107
rotary, 107
-tests for heterophoria, 103, 104, 105, 106, 107
for strabismus, 673
Prisms, exercises with, 680
numbering of, 688
prescription of, in heterophoria, 680
in myopia, 716
Probe, Bowman's, 235
Caldwell's lacrimal, 237
false passage by a, 236
Snellen's, 229, 235
Theobald's, 229
Probing of nasal duct, 235
Projection of false image, 657
Prolapse of iris, 395, 454
of vitreous humor, 452
Prosthesis, ocular, 613
Protargol, 251, 255, 257, 290
Protoplasmic stains, 782
Protrusion of globe, 74
Pseudobulbar paralysis, 742
Pseudo-glioma, 480
Pseudo-neuroepithelioma, 480
Pseudonterygium, 287
Psorophthalmia, 185
Pterygium, 285
actual cautery in treatment of, 297
cicatricial, 287
congenital, 239
crassum, 285
etiology of, 286
excision of, 297
McReynolds's operation for, 297
operations for, 297
pathology of, 286
prognosis of, 286
tenue, 285
transplantation of, 297
treatment of, 286
Ptosis, 191, 733
adiposa, 196
congenital, 159
operations for, 196
spurious, 191
treatment of, 191
unilateral, 191
Pulsating exophthalmos, 638
Pulsation of retinal arteries, 573
vessels in glaucoma, 572, 573
Puncta lacrimalia, acquired anomalies of, 222
Punctate conditions of the fundus, 481
proximum. See *Near point.*
remotum. See *Far point.*
Pupil, Argyll Robertson, 97
contraction of, 71
from irritation, 96
from paralysis, 96
dilation of, 71
paralytic, 71
spastic, 71
examination of, 95
Gifford-Galassi reflex of, 98
Haab's reflex of, 98
measurement of, 96
occlusion of, 387
Robertson, 97
skin-reflex of, 98
Wernicke's inaction of, 97
Westphal-Piltz reflex of, 98
Pupillary area, examination of, 98
reaction, facts concerning, 97
Pupillometer, 96
PURKINJE-SANSON images, 67
Purulent chorioiditis, 429
conjunctivitis, 262
cyclitis, 409
ophthalmia, 262
PUSEY, B., 379
Pustular conjunctivitis, 254
PYLE, 135, 468
Pyoktanin, 227

QUAGLINO, 361, 591
QUÉNU, 584
QUINCKE'S reaction, 790
Quinin-amaurosis, 543

RABL, 4
Radian, 688
RAEHLMANN, 225, 281, 326, 345, 523
RAMSAY, 188, 191, 323, 615
RANDALL, 135
RANDOLPH, R. L., 239, 262, 471, 601
Raupenhaar-ophthalmie, 285
RECKLINGHAUSEN, 338
RÉCLUS, 584
Rectus, external. See *External rectus.*
Recurrent third-nerve paralysis, 667
Red-blindness, 550
Reduced eye of Listing, 65
Refraction, 62
errors of, 686
REIK, H. O., 438
REISINGER, 360
Relapsing herpes of the cornea, 333
Relative ocular movements, 74
Renal vascular retinitis, 505
Renversement of a corneal flap, 358
Retina, anatomy of, 42
anemia of, 489

Retina, anesthesia of, 526
aneurism of vessels of, 492
angioid streaks in, 518
apoplexy of, 492
cerebral layers of, 44
ciliary and iridal parts of, 46
coloboma of, 481
concussion of, 521
congenital anomalies of, 481
cysts of, 482
detachment of, 416, 522
diagnosis of, 524
etiology of, 522
pathology of, 523
prognosis of, 524
symptoms of, 523
treatment of, 525
development of, 6
diseases of, 481-527
edematous swelling of, 521
embolism of central artery of, 494
diagnosis of, 497
etiology of, 496
pathology of, 496
symptoms of, 495
treatment of, 497
functional diseases of, 526
functions of, 77
glioma of, 482
hemorrhages in, 492
hyperemia of, 489
active, 490
passive, 490
hyperesthesia of, 526
inflammation of, 500
irritation of, 526
ischemia of, 489
layers of, 43
light-streak of vessels of, 58
neuroepithelial layer of, 46
neuroepithelioma of, 418, 482
opaque nerve-fibres in, 481
optic part of, 43
pigmentary degeneration of, 519
pseudo-neuroepithelioma of, 487
pulsation of vessels of, 58, 569, 572, 573
punctate conditions of, 515
Purkinje's phantom image of vessels of, 79
rupture of, 521
subhyaloid hemorrhage of, 494
traumatic anesthesia of, 522
edema of, 521
perforations of macula of, 522
tuberculosis of, 527
Retinal artery, embolism of, 494
thrombosis of, 498
hemorrhage, 521
image, demonstration of, 65
formation of, 64
size of, 66
pigmentation, variations in, 481
vasculitis, 490
vein, thrombosis of, 498
vessels, sclerosis of, 490
Retinitis, albuminuric, 505
diagnosis of, 508
etiology of, 505
induction of premature labor in, 510, 511
of pregnancy, 509
pathology of, 506
prognosis in, 509
statistics of, 505
symptoms of, 506
treatment of, 511
Bright's, 505
circinata, 513
diabetic, 503
central punctate, 503, 504
embolic, 516
from concussion, 500
from excessive light, 514
glycosuric, 503
gouty, 511
gravidarum, 505, 510
hemorrhagic, 492
hyperplastic proliferating, 517
leucocythemic, 503
nyctalopic, 500
parenchymatous, 502
pigmentosa, 519
Retinitis pigmentosa, diagnosis of, 520
etiology of, 519
ophthalmoscopic signs of, 519
pathology of, 520
prognosis of, 520
symptoms of, 519
treatment of, 521
proliferans, 480, 517
proliferating, 517
punctata albescens, 515
purulent, 516
metastatic, 516
traumatic, 516
renal vascular, 505
saturnine, 508
septic, 517
serous, 500
simple, 500
solar, 514
specific, 501
striata, 518
syphilitic, 500, 501
prognosis in, 502
treatment of, 502
traumatic, 521
Retinoscopy, 145-151
Retraction of the globe, 74
Retrobulbar optic neuritis, 539
Retrotarsal folds, removal of, 299
REVERDIN, 213
Reversal of color-fields, 527
REYMOND, 315
Rhagades, 161
Ribbon-shaped corneal opacity, 334
RICHEY, 578
RICHTER, 460
RIDLEY, 277
Ring ulcer of cornea, 310
RISLEY, 107, 459, 578
RIVERS, 515
ROBERTSON, ARGYLL, operation for ectropion, 208, 209
symptom, 97, 741
Rodent ulcer of cornea, 310
of eyelids, 176, 177
Rods and cones in vision, 79
ROEMER, 433
ROENTGEN rays. See *X-rays*.
ROGMAN, 361
ROSENBACH'S phenomenon, 644
ROSENSTEIN, 509
ROSER, 311
Rotary prism, 79
ROTH, 516, 517
Round central ulcer of cornea, 310
Rubeola, 184
RUMSCHEWITZ, 305
Rupture of choroid, 184
Schlemm's canal, 369
RUSSELL, 513
RYDEL, 522

SACHS, 498, 512
SAEMISCH, 311, 315, 316, 357, 361
incision in hypopyon keratitis, 315, 357
SAENGER, 642, 643, 644
Safranin, 781
Salicylate of sodium in phlyctenular conjunctivitis, 274
in sympathetic ophthalmitis, 605
"Salmon patches," 323
SALZER, 222
SANTORINI, emissary vein of, 636
Sarcoma of caruncle, 296
of choroid, 415
of ciliary body, 405
of conjunctiva, 244
of cornea, 305
of eyelid, 177
of iris, 378
of lacrimal gland, 221
of optic nerve, 531
of orbit, 624
SATTLER, 277, 281, 289
Saturnine retinitis, 508
SAVAGE'S orthoptic exercises in heterophoria, 681
Scarlatina, 184
SCARPA, 367, 368
SCHAPRINGER, 641

SCHEFFELS, 502
SCHEINER'S experiment, 72
SCHIECK, 305
SCHIELE, 283
SCHIESS, 437
SCHIRMER, 365, 456, 594, 596, 598, 599, 601
SCHLEMM'S canal, 41
  rupture of, 369
SCHLESINGER, 506
SCHMIDT, A., 220
SCHMIDT-RIMPLER, 603
SCHNABEL, 425
SCHÖBL, 495, 503, 504, 518, 524
SCHOEN, 67
SCHREIBER, 275
SCHROEDER, 224
SCHWALBE, 41
SCHWEIGGER, 306, 461, 586
Sclera, anatomy of, 32
  congenital anomalies of, 361
  diseases of, 361
  ectasiæ of, 366
  examination of, 92
  foreign bodies in, 370
  in fundus picture, 61
  injuries of, 368
    treatment of, 369
  melanosis of, 361
  operations upon, 373
  osteoma of, 361
  perforating wounds of, 368
  perforation of, during tenotomy, 682
  pigmentation of, 361
  protrusions of, 366
  rupture of, 368, 369
  telangiectasis of, 361
  tumors of, 361
  wounds of, 368
Scleral puncture in detachment of retina, 525
  staphylomata, 366
  wounds, infected, cauterization in, 370
Scleritis, 364
  deep, 364
  superficial, 361
  treatment of, 366
Sclerocyclocotomy, 590
Sclerokerato-iritis, 364
Scleronyxis, 462
Sclero-optic neurectomy, 611
Sclerosing keratitis, 364
Sclerosis of corneal limbus, 340
Sclerotic, large paracentesis of, 590
Sclerotica. See *Sclera*.
Sclerotomy, 591
  anterior, 591
  in detachment of retina, 525
  in glaucoma, 591
  posterior, 593
Scopolamin, 132
Scotoma, central, 126
  false, 126
  negative, 126
  positive, 126
  scintillans, 557
  true, 126
SCOTT, 226
Scrofulous keratitis, 320
  ophthalmia, 272
SEABROOK, 283
Seborrhea, 180, 185
  of eyelids, 180
  of palpebral margins, 185
Second principal focus, 62
"Second sight," 440
Secondary deviation, 657
  eversion of eyelid, 89
  eye-cup and eye-membranes, 4
  positions of eyes, 73
SECONDI, 315
SEEBECK, 128
SEELEY, 351
Senile iritis, 389
SENN, 618
Serous cyclitis, 333, 388, 406
  iritis, 333, 384
Serpiginous syphilide of eyelids, 173
  ulcer of cornea, 311
SESEMANN, 536
Shadow test, 145-151
SHARKEY, 643
SHERER, 544
SHOEMAKER, W. T., chapter contributed by, 616
SHUMWAY, 487
SICHEL, 525
Siderophone, 157
Sideroscope, 153
Siderosis of conjunctiva, 290
  of cornea, 341
  of eyelids, 167
SIEGRIST, 498
SILCOCK, 419
SILEX, 239, 305, 507, 509, 510
SILVESTRI, 307
SIMON, R., 305
Simple conjunctivitis, 251
  granular conjunctivitis, 257
  hypertrophy of lacrimal gland, 220
  keratomalacia, 318
  optical system, 62
  ulcer of the cornea, 310
Simulated amblyopia, 556
  blindness, tests for, 151
Sinus, cavernous, thrombosis of, 636
Sinuses, affections of, 633
Sixth nerve, 19
  radiations of, 51
Skiascopy, 145
  in astigmatism, 149
  in emmetropia, 147
  in hypermetropia, 147
  in myopia, 147, 149
  with concave mirror, 147
  with plane mirror, 150
Skin diseases of eyelids, 160
Skin-grafting, Reverdin's method of, 217
  Thiersch's method of, 217
  without a pedicle, 216
Skin-transplantation. See *Blepharoplasty*.
Small-pox. See *Variola*.
SMITH, EUGENE, 341, 394, 400, 459, 460
SMITH, PRIESTLEY, 51, 93, 113, 125, 497, 562, 563, 773
SNELL, A. C., 378
SNELL, S., 135, 242
SNELLEN, 119, 120, 151, 199, 203, 204, 207, 235, 304, 316, 345, 583
  operation for ectropion, 207
    for entropion, 203
  test-types of, 119
Snow-blindness, 514
Solid edema of eyelids, 169
Solitary tubercle of chorioid, 418
SOURDILLE, 269, 538
Spasm of accommodation, 697
  of ocular muscles, 669
  of orbicularis, 189
Spastic ectropion, 280, 457
  enophthalmos, 640
  strabismus, 669
Specific keratitis, 321
Spectacles, care of, 771
Spectral colors, 128
Sphenoidal fissure, 9
  sinus, diseases of, 633
Spheric aberration, 69
  lenses, 688
Sphincter of the iris, 34
Sphincterectomy, 399
Spongy exudation into anterior chamber, 457
Spongy iritis, 385
Spontaneous gangrene of eyelids, 179
Spontaneous rupture of eyeball, 418
Spring conjunctivitis, 260
Spurious optic neuritis, 530, 541
Squint. See *Strabismus*.
Staining eye sections, 779
STANDISH, MYLES, 268
Staphylectomy, 356
Staphylococci in corneal ulcers, 309
Staphyloma, 306, 309
  anterior, 341
  congenital, of cornea, 304
  of cornea, 306, 309, 341
    treatment of, 343
  of sclera, 366
    treatment of, 368
  operations for, 356
  pellucidum, 344
  posterior, 367, 425, 711

STARKLE, 525
Stauungspapille, 533
Steatoma, 180
STEINER, 280
STELLWAG, 216, 304
  sign in exophthalmic goitre, 613
Stenopeic slit, 721
  spectacles, 345
STEPHENSON, 225, 255, 257, 258, 281, 284, 290, 300, 327, 521
Stereoscope, 674
Stereoscopic tests, 152
  vision, 76
Sterilization of dressings, 754
  of instruments, 753
STERNBERG, 251
STEVENS'S classification of heterophorias, 101
  clinoscope, 112
  method of tenotomy, 683
  phorometer, 107
  tropometer, 110
STIEREN, 406
STILLING, 234
STILLSON, 525
STIRLING, 578
STÖLTING, 583
STORY, 527, 605
Strabismometer, 115
Strabismometry, 112
Strabismus, 670
  alternating, 671
  amblyopia in, 671
  angle of, 672
  angular method of measurement of, 672
  comitant convergent, 671
    divergent, 671
  convergent, 671
    causes of, 671
    educative treatment of, 674
    in hypermetropia, 698
    occlusion of fixing eye in, 674
    operative treatment of, 675
    orthoptic training of, 674
    spectacle treatment of, 673
    tenotomy in, 675
    treatment of, 674
      results of tenotomy in, 675
  deorsumvergent, 670
  divergent, 670
  concomitant, 671
    causes of, 672
    operations for, 681
    tenotomy in, 675
    treatment of, 673
  etiology of, 671
  in myopia, 714
  latent. See *Heterophoria.*
  measurement of, 112, 672
  monocular, 671
  operative correction of, 675, 681
  orthoptic treatment of, 674
  paralytic, 657
    diagnosis of affected eye in, 661
    diplopia in, 657
    etiology of, 664
    field of fixation in, 658
    from injury, 667
    general symptoms of, 657
    method of examination of eye in, 663
    of basal origin, 667
    of central origin, 664
    of nuclear origin, 665
    of peripheral origin, 664
    orbital, 667
    position of false image, 657
      of head in, 659
    primary deviation in, 657
    prognosis of, 668
    secondary deviation in, 657
    special symptoms of, 661
    table of diagnostic symptoms of, 661, 662, 663
    treatment of, 668
  periodic, 671
  permanent alternating, 671
    monocular, 671
  phenomena of vision in, 671
  results of tenotomy, 675
  spastic, 669
  sursumvergent, 670
Strabismus, treatment of, 673
  varieties of, 670
STREATFEILD, 221
Streptococci, in corneal ulcers, 309
Striate keratitis, 338
  retinitis, 518
Striped keratitis, 338
STROSCHEIN flask, 247, 250
Strumous keratitis, 320, 321
  ophthalmia, 272
Strychnin in heterophoria, 680
  in ocular paralysis, 666, 668
  in optic-nerve atrophy, 547
STRZEMINSKI, 182, 267
Stye, 166
Style, lacrimal, 230
Subacute conjunctivitis, 256
Subconjunctival cysts, 242
  ecchymosis, 293
  hemorrhage, 293
  injections, 301, 455
  lipoma, 240
Subhyaloid hemorrhage, 494
Subjective sensations of light. See *Photopsia.*
Subretinal cysticercus, 488
  echinococcus, 489
Substantia propria, 30
Suction operation for cataract, 460
Sudamina, 179
SUKER, 351, 462
Sulphate of copper, 262, 283
Superficial punctate keratitis, 333
  scleritis, 361
Superior oblique, paralysis of, 662
  rectus, paralysis of, 662
Suppuration after cataract extraction, 455
Suppurative chorioiditis, 429
  dacryocystitis, 226
  iritis, 385
  keratitis, 307
Suprarenal capsule, extract of, 755
Surface needling of cornea, 351
Sursumduction, 109
Sursumvergence, 109
Suspensory ligament of lens, 54
SÜSSKIND, 222
SUTPHEN, 525
SWANZY, 319, 336, 496, 514
SWEET, 154, 371
  method with x-rays, 154
Sycosis tarsi, 185
Symblepharon, 158, 190, 218, 293, 295
  operations for Arlt's, 298
    Harlan's, 299
    Knapp's, 298
    Teale's, 298
  skin grafts in, 299
  transplantation of rabbit's conjunctiva in, 298
  treatment of, 298
Sympathetic affections, conditions producing, 594
  eye diseases, 594
  fibrinous uveitis, 599
  ganglion, superior cervical, excision of, 586
  inflammation. See *Sympathetic ophthalmitis.*
  irritation, 594, 595
    causes of, 594
    diagnosis of, 596
    prognosis of, 597
    symptoms of, 595
    treatment of, 597
  nerve, changes in, in glaucoma, 565
  ophthalmitis, 458, 598
    bacteria theories of, 603
    causes of, 598
    ciliary-nerve theory of, 603
    combined theories of, 603
    enucleation in, 604
    exenteration (evisceration) in, 604
    following cataract extraction, 458
    immunity to, 613
    optic-nerve theory of, 603
    pathogenesis of, 602
    pathology of, 601
    period of latency in, 598
    prognosis of, 601
    prophylaxis of, 604
    symptoms of, 598

Sympathetic ophthalmitis, toxin theories of, 603
treatment of, 604
papilloretinitis, 601
Sympatheticectomy, 586
Sympathizing eye, 594
Synechia, corporis vitrei, 477
étincelant, 477
scintillans, 477
sparkling, 477
Syndectomy, 301
Synechia, annular posterior, 387
anterior, 309
division of, 393
posterior, 387
Syphilis, 418, 509
in chronic ophthalmoplegia, 666
of conjunctiva, 291
of eyelids, 173
of lacrimal gland, 221
Syphilitic arteritis of the retina, 501
chorioidoretinitis, 426
hemorrhagic retinitis, 501
lesions of conjunctiva, 291
perivasculitis of the retina, 501
retinitis, 500, 501
teeth, 323
Syringomyelia, 744
Syrup of iodid of iron, 274
SZOKALSKI, 305
SZYMANOWSKI, 215

Tabes dorsalis, 741
TAGLIACOZZI, 215
TALAMON, 251
TANSLEY, 197
operation for ptosis, 197
Tapetum, 39
Tarsal plate, 26
tumor, 181
Tarsitis, 162
Tarsorrhaphy, 188, 200
operations, 200
Tarsus, 26
inflammation of, 162
removal of, 299
Tattooing, accidental, of skin, 167
of cornea, 351, 359
TAY WAREN, 424, 512
chorioiditis of, 426
TEALE operation for symblepharon, 298
Tear-stone, 223
Teeth, notched, 323
pegged, 323
Teichopsia, 557
Tendo oculi, 26
Tenonitis, 632
after tenotomy, 682
TENON'S capsule, 11
advancement of, 685
Tenotomy, 681
accidents during, 682
complete, 681
complications after, 682
graduated, 683
in heterophoria, 683
in convergent concomitant strabismus, 675
in divergence excess, 681
in heterophoria, 681
in myopia, 716
in strabismus, 681
Tension, intra-ocular, 99, 562
determination of, by manometer, 100
effect of irritation of fifth nerve on, 562
of mydriatics on, 132
of sympathetic nerve on, 562
examination of, 99
in cataract, 446
in detachment of retina, 523
in glaucoma, 562
normal intra-ocular, 562
periodic variations of, in glaucoma, 572
Tensor chorioidea, 38
TEPLJASCHIN, 173
TERSON, 359
Test-types, Burchardt's, 121
Monoyer's, 120
Snellen's, 118
Wallace's, 119
Tests for simulated blindness, 151
Tetany, 743
Thalamus opticus, 41, 736
THEOBALD, probes of, 229, 235
THEODOR, DUKE CARL, 507
Therapeutic iridectomy, 398
THIERSCH, 211, 213, 217, 299
method of skin-grafting, 217
Thiosinamin, 351
Third nerve,
radiations of, 51
recurrent paralysis of, 667
THOMAS, 496
THOMPSON, J. H., 255, 283
THOMSEN'S disease, 744
THOMSON, 128
Thrombosis of cavernous sinus, 636
of central retinal artery, 498
vein, 498
of orbital vein, 498
TILLEY, 633
Tinea tarsi, 185
Tobacco amblyopia, 542
TODD, 611
Tonometers, 100
Tonsure of the cornea, 301
Toric lens, 690
TORNATOLA
Torsion, 565
Total prolapse of iris, 309
staphyloma of cornea, 342
Trachoma, 277
acute, 279
bodies, 277, 279
canthoplasty in, 200, 201
canthotomy in, 200
chronic, 278
coccus, 277
complications of, 280
condition favoring, 278
contagiousness of, 278
copper in treatment of, 283
corneal opacities after, 280
definition of, 277
diagnosis of, 281
etiology of, 277
excision in, 283, 299
expression in, 283, 300
follicles, 279
follicular, 257
forceps, Knapp's roller, 300
forms of, 278
geographic limitations of, 278
granular, 278
grattage in, 283, 301
ichthyol in treatment of, 283
iodic acid in, 283
iodin in treatment of, 283
Jequirity in, 284
microorganisms in, 277
mixed, 280
papillary, 278
pathology of, 281
periectomy in, 284, 301
peritomy in, 301
prognosis of, 282
prophylaxis of, 282
racial susceptibility to, 278
sequelæ of, 280
silver in treatment of, 283
strong bichlorid solutions in, 301
treatment of, 282
vegetable fungus in, 277
Transfixion of the iris, 400
Transplantation of skin without a pedicle, 216
Transverse film of the cornea, 334
Traumatic cataract, 445, 464
irideremia, 395
retinitis, 521
Tremulous iris, 396
Trephining of the cornea, 360
Trial case, 772
-frame, 120
Trichiasis, 190, 201, 280
after trachoma, 280
operations for, 201
treatment of, 201
Trichosis caruncuræ, 296
TRIPIER, 230
TRONCOSO, 569, 570
Tropacocain, 754
Tropometer, 110

Tropometer, use of, 111
TROUSSEAU, 359
TRUC, 131
TSCHERNING, 66, 550
Tubercular disease of conjunctiva, 289
  mass, large, 413
  meningitis, 740
Tuberculin injections, 380
Tuberculosis of choroid, 412
  of conjunctiva, 289
  of cornea, 341
  of eyelids, 172
  of iris, 380
  of retina, 527
Tumors, intra-ocular, 377, 404, 414, 482
  of brain, 508
  of caruncle, 296
  of cerebellum, 534
  of choroid, 414
  of iris, 377
  of lacrimal gland, 221
  of optic nerve, 530
  of orbit, 618
Tunica vasculosa lentis, 3
TWEEDY, 641
Tylosis, 186
TYRRELL, drilling operation of, 461, 600, 605

UHTHOFF, 303, 307, 504, 505, 555
Ulcer, Jacob's, 177
  of conjunctiva, 292
  of cornea, 308, 309
  rodent, 176, 177
Ulcus corneæ serpens, 311
ULRICH, 554, 555
Upper cul-de-sac, excision of, 299
Uratic conjunctivitis, 276
Uremic amaurosis, 552
Uridrosis, 179
Urticaria, 160
Use of miotics, 134
  mydriatics, 131, 132
Uveal tract, 33
Uvélte irienne, 390
Uveitis, anterior, 364
  fibrinous, 599
  serosa, 599

Vaccinia of conjunctiva, 284
  of eyelids, 184
VAIL, D. T., 539
Validol, 558
VALUDE, 131, 334
VAN BAMBEKE, 2
VAN FLEET, 197
VAN GIESON'S stain, 782
Varicella of conjunctiva, 284
  of eyelids, 184
Varicose veins of eyelids, 170
Variola, 183, 276, 313
Variolar conjunctivitis, 276
  ulcers of cornea, 313
Vascular growths in iris, 380
  keratitis, 325
  systems of the eye, 90
Vasculitis, 490
VEASEY, 177, 178, 252, 379
Veins of eyelids, 25
  of iris, 35
  of orbit, 21
Venæ vorticosæ, 39
  in glaucoma, 567
Venous pulsation, 59
Veratrin ointment, 190
VERDESE, 307
Vernal conjunctivitis, 260
Verruca of eyelid, 168
Verrucosities of optic-nerve head, 532
Vertical meridian, 768
Vertigo, 659
Vesicular catarrh, 254
VINCENTIIS, 225, 281, 291
VIRCHOW, 429
Vision abnormalities of field of, 736
  binocular, 75
  clearness of, 81
  direct and indirect, 79
  field of, 77
    false projection of field of, in paralytic strabismus, 657
Vision, field of, method of testing, 124
    Bjerrum's method of ascertaining field of, 125
    color-field of, 78
  in glaucoma, 569
  in hysteric amblyopia, 558
  in optic-nerve atrophy, 545
  in retinal anesthesia, 527
  for colors, 78
  iridescent, in glaucoma, 567
  method of ascertaining field of, 124
  normal acuteness of, 118
  rods and cones in, 79
  testing acuteness of, 118
Visual acuity, 65
  testing of, 118
  amnesia, 739
  angle, 65, 119
  aphasia, 739
  area, 41
    diagrams showing, 42
  centre, 82
  field, 77
  hallucinations, 739
  line, 68
    rotation of eyeball around, 73, 656
  purple, 80
  radiations, 82
  rods and cones, 79
  sensations, duration of, 80
  tract, 735
Vitiligo, 170
Vitreous body or humor, anatomy of, 54
  animal parasites in, 478
  artificial, 611
  blood-vessel formation in, 479
  cholesterin in, 477
  coloboma of, 473
  corpuscles of, 54
  cysticercus of, 478, 487
  degeneration of, in myopia, 710
  detachment of, 480
  development of, 3
  diseases of, 473-480
  dust-like opacities of, 475
  entozoa in, 478
  examination of, 99
  fibrillæ of, 54
  filaria sanguinis in, 478
  fluidity of, 477
  foreign bodies in, 480
  formation of new vessels in, 479
  hemorrhage into, 476
  hyaloid membrane of, 54
  inflammation of, 473
  loss of, 453
  membranes in, 480
  opacities of, 475
    causes of, 475
    in chorioiditis, 422
    treatment of, 476
  parasites in, 478
  prolapse of, 453
  purulent inflammation of, 474
  spontaneous hemorrhage into, 476
  spontaneous inflammation of, 474
  suppurative inflammation of, 474
VOLKMANN, 301
VON AMMON, 201, 206, 208, 367
VON GRAEFE, 380, 436, 525, 584
VON HIPPEL, 158, 360
VON HÖLDER, 649
VON KÖLLIKER, illustrations from, 2, 5
VON MICHEL, 277, 324, 380, 498
VOSSIUS, 339
VROLIK, 471

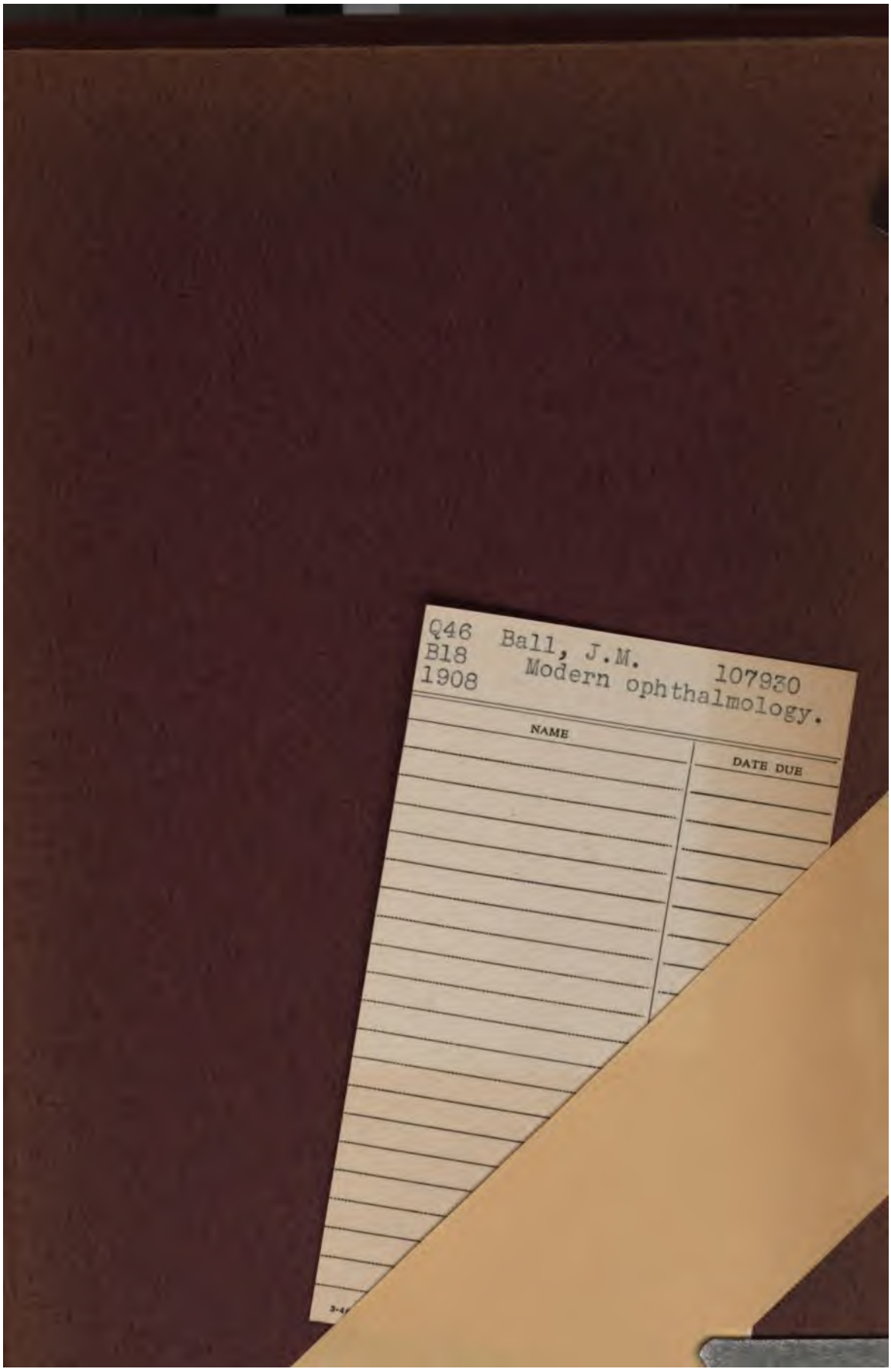
WAGENMANN, 324
WAGNER, 478, 505
WALLACE, test-type of, 119
WALTER, 522
WALZBERG, illustration from, 171
WARBURG'S tincture, 392
WARDROP, 240
Washing of eye specimens, 775
Watch-glass in purulent conjunctivitis, 265
WATSON, 361
WEBER, 234, 307, 311
WEEKS, 252, 339, 508
  bacillus, 256

WEGNER, 562
WEHRLE, 312, 315
WEICHSELBAUM, 251
WEIDLER, 230
WEIGERT'S stain, 783
WEINBAUM, 578
WENZEL, 461
WERNICKE'S pupillary symptom, 97, 739
WERNKE, 225
WESTCOTT, C. D., 305
WESTPHAL-PILTZ reaction, 98
WHITE, 467
WICHERKIEWICZ, 207, 330
WIENER, 291
WILBRAND, 642, 643, 644
WILDER'S operation for ptosis, 196
WILLIAMS, 128
WILSON, 484
WINSELMANN, 525
WINTERSTEINER, 482, 485
WOLFE, 526
WOLFF, 199, 471
WOLFFBERG, test-type of, 121
WOLFNER, 330
WOLLENBERG, 528
WOOD, C. A., 379, 430
WOODS, H., 427
WOOLHOUSE, 400
Word-blindness, 739
WORTH, C., 674
Wounds and injuries of chorioid, 433
  of ciliary body, 409
  of conjunctiva, 294
  of cornea, 351
  of crystalline lens, 471
  of eyeball, 370
  of eyelids, 293
  of iris, 394
Wounds and injuries of optic nerve, 560
  of orbit, 646
  of retina, 521
  of sclera, 368
WRIGHT, R. A., 283
WURDEMANN, 304

Xanthelasma of the cornea, 341
Xanthoma, 172
Xanthopsia, 556, 559
Xeroderma pigmentosum, 171
Xeroform, 262
Xerophthalmos, 288
Xerosis, 280, 288
  bacillus, 269, 271
  epithelial, 288
  of conjunctiva, 288, 318
  parenchymatous, 288
Xerotic keratitis, 318
X-rays in detecting foreign bodies, 153
  in epithelioma of eyelids, 177
  in granular conjunctivitis, 283
  in tuberculosis of conjunctiva, 290

Yaws, 161
Yellow ointment in blepharitis, 187
  oxid of mercury, 187, 274, 321
  spot, 42
YOUNG'S theory of color-perception, 551

ZEHENDER, 347
ZEISS'S glands, 166
ZELLWEGER, 378
ZENTMAYER, W., chapter contributed by, 654
ZIEGLER, 555
ZIEM, 278
Zonula of Zinn, anatomy of, 54
Zonular opacity of the cornea, 334


Q46 Ball, J.M. 107930
B18 Modern ophthalmology.
1908
NAME
DATE DUE